THE Journal Kansas Medical Society

JULY
1961

VOL LXII
NO VII

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inside as well as outside the hospital...
staphylococci usually remain sensitive to

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(chloramphenicol, Parke-Davis)

That the sensitivity patterns of "street" staphylococci differ widely from those of "hospital" staphylococci is a well-established clinical fact.[1-5] Although strains of staphylococci encountered in general practice have remained relatively sensitive to a number of antibiotics,[6] the problem of antibiotic-resistant staphylococci appears to be a threat to all patients in hospitals today. It is encouraging to note, however, "...that a relatively small percentage of strains develop resistance to chloramphenicol, despite the consumption of large amounts of this antibiotic."[7]

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These strains of coagulase-positive staphylococci were isolated from hospitalized patients at a large county hospital during the year 1959. Sensitivity tests were done by the disc method.

*Adapted from Bauer, Perry, & Kirby[1]

References: (1) Bauer, A. W.; Perry, D. M., & Kirby, W. M. M.: *J.A.M.A.* **173**:475, 1960. (2) Fisher, M. W.: *Arch. Int. Med.* **105**:413, 1960. (3) Cohen, S.: *Circulation* **20**:96, 1959. (4) Edwards, T. S.: *Am. J. Ophth.* **48**, Part II:19, 1959. (5) Smith, I. M.: Staphylococcal Infections, Chicago, The Year Book Publishers, Inc., 1958, p. 148. (6) Petersdorf, R. G.; Rose, M. C.; Minchew, H. B.; Keene, W. R., & Bennett, I. L., Jr.: *Arch. Int. Med.* **105**:398, 1960. (7) Editorial: *J.A.M.A.* **173**:544, 1960. (8) Finland, M.; Jones, W. F., Jr., & Bennett, I. L., Jr.: *Arch. Int. Med.* **104**:365, 1959.

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Volume LXII, Number 7—July, 1961

The JOURNAL of the KANSAS MEDICAL SOCIETY

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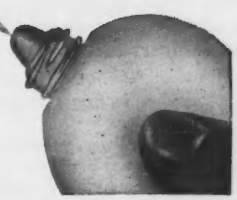

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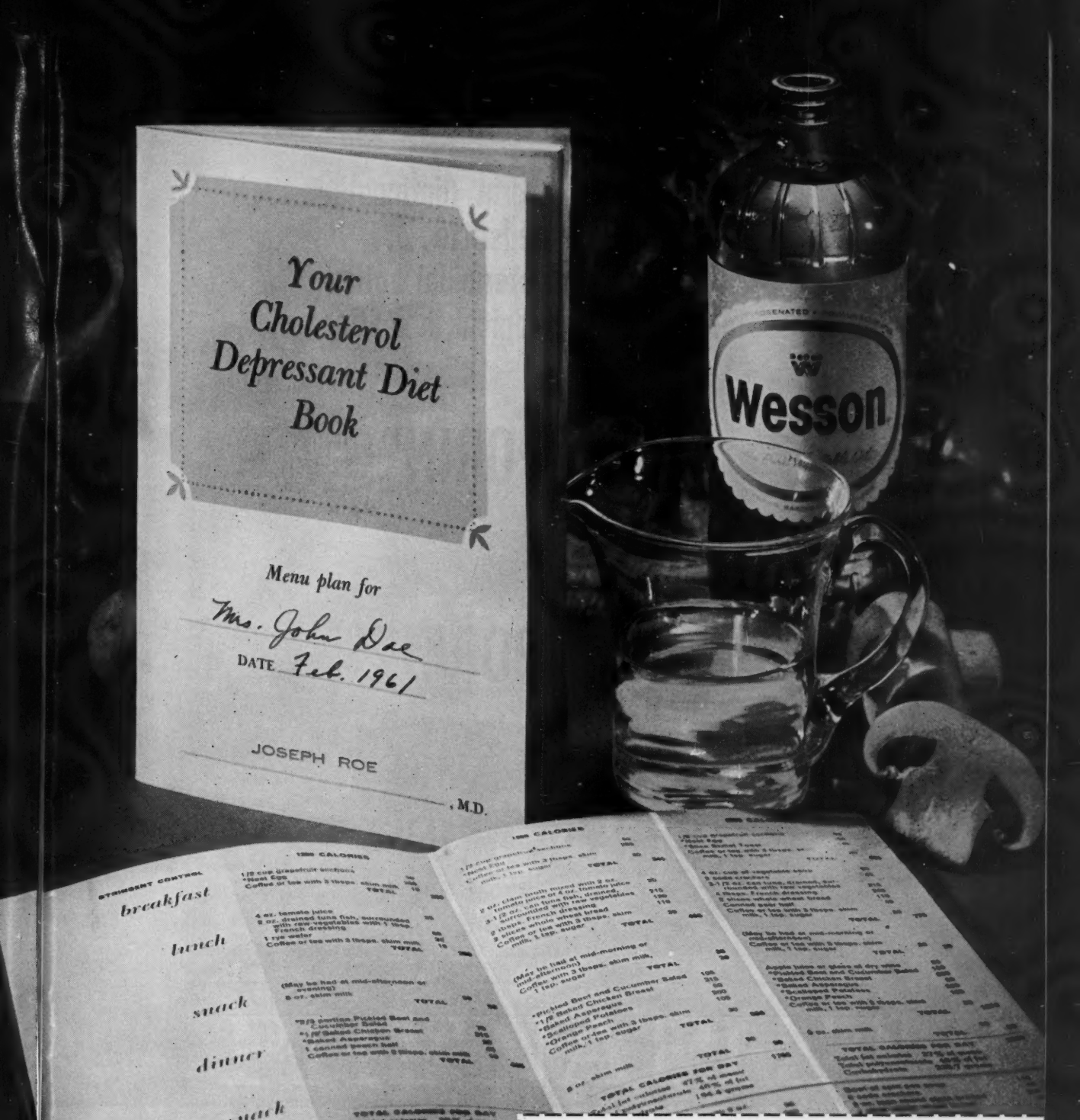

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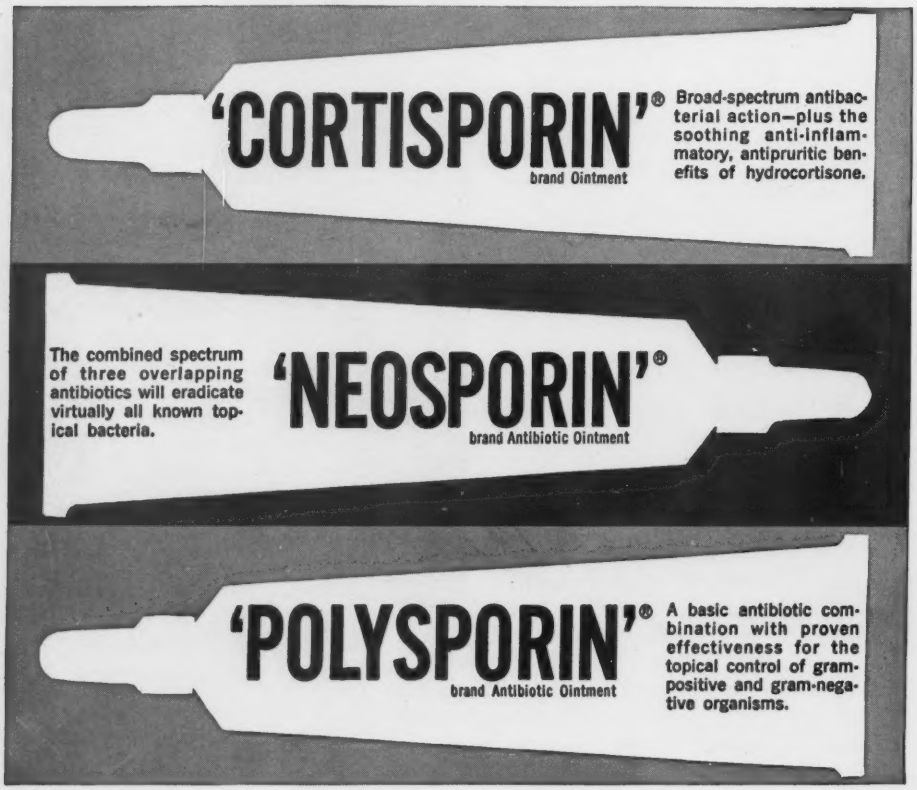
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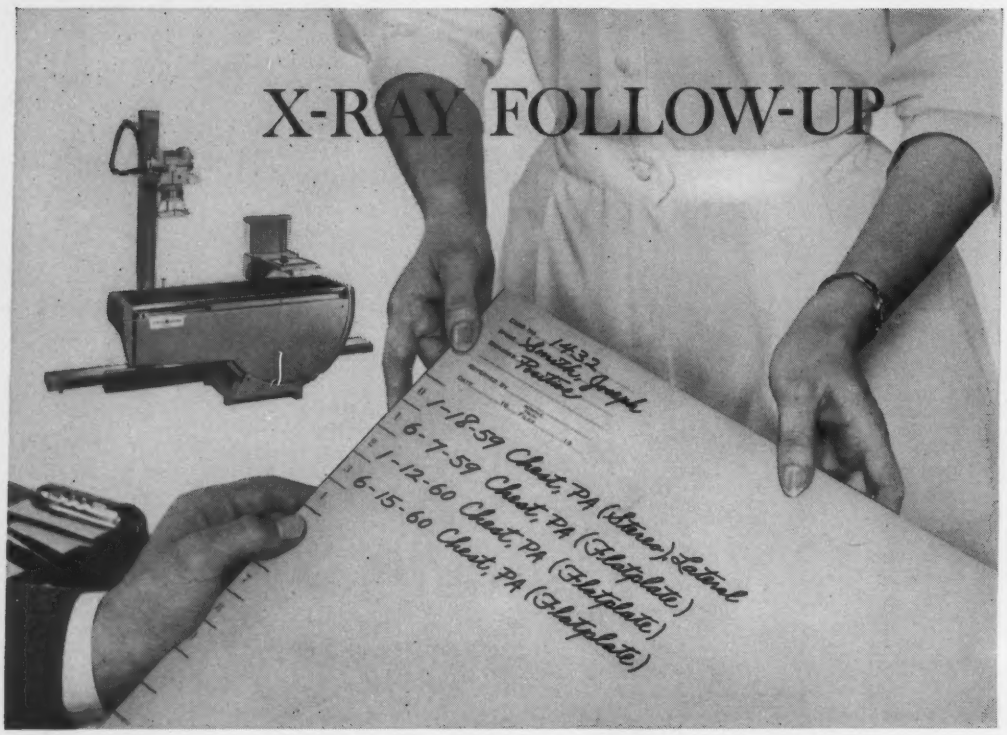

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References: 1. Garber, R. C.: J. Florida M. A. *45*:549 (Nov.) 1958. 2. Lipton, M. I.: Pennsylvania M. J. *64*:60 (Jan.) 1961. 3. Ayd, F. J., Jr.: Psychotropic Drugs, S. Garattini and V. Ghetti, eds., New York, Elsevier Publishing Co., 1957, p. 548. 4. McGettigan, D. L.: West. Med. *1*:8 (Jan.) 1960.

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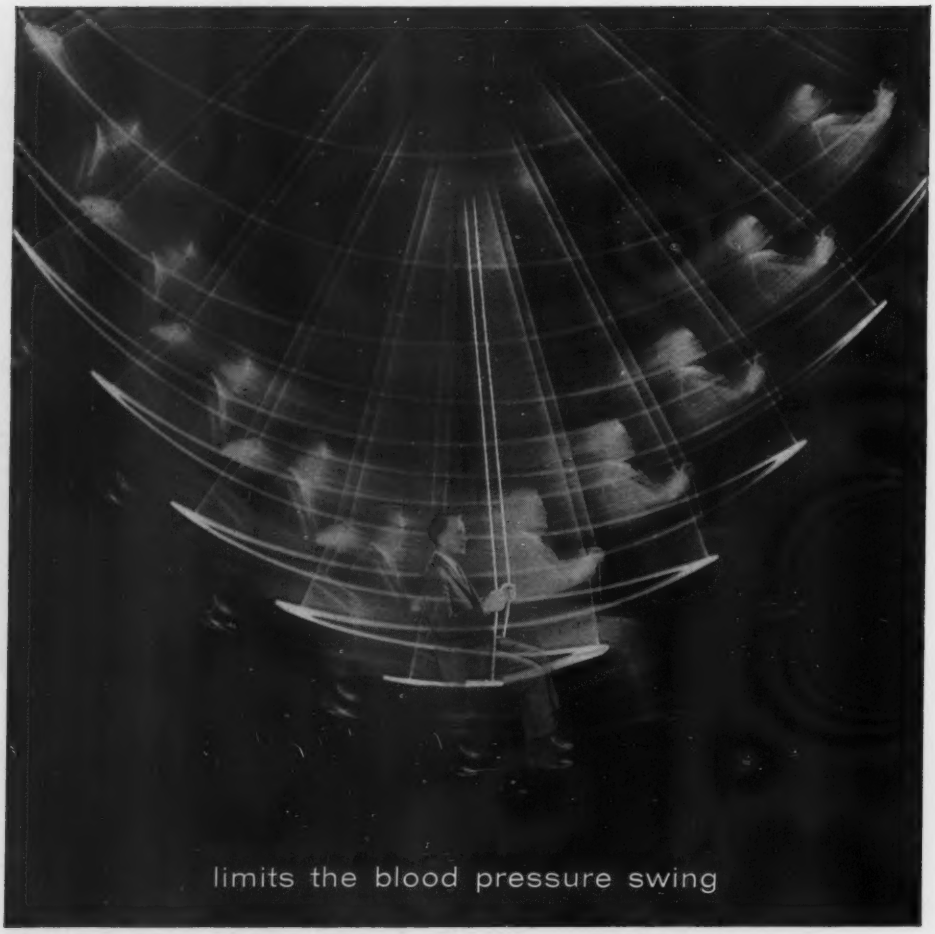

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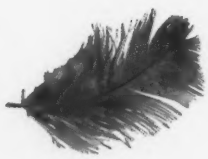

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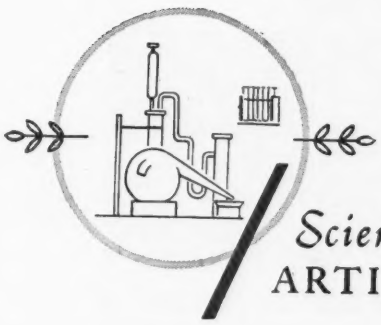

F.A.C.P. Regional Meeting

Abstracts of papers presented at the Regional Meeting of American College of Physicians at Topeka on March 3, 1961

THE TRADITIONAL CONCEPT OF MENTAL DISEASE

W. C. Goodpasture, M.D., Wichita

Traditionally Man has felt that insights into life are necessary if he is to escape despair, that life will catch up with him unless he learns to live it in the right way, in a word he will become mentally ill. The insights necessary seem to me to be as follows:

1. Personal denial is a reality. Knowledge works but is in short supply. Power is tempting but is no substitute for knowledge. Can't force what is not known.

2. Awareness of possible personal denial without knowledge of how to meet it is responsible for the nervous crisis.

3. The nervous crisis can be met in two ways.

a. Refusal to accept personal denial—more and more frantic appeasing and defying, hoping that power can win for him, becoming mentally sicker and sicker until tragedy befalls him unless he can stop and—

b. Accept personal denial—patiently continuing to put hope in acquiring knowledge, remaining stable and mentally healthy.

4. Understanding the nature of the person helps make the correct choice when confronted with a nervous crisis.

a. Personal wishes are only personal, never correct, never incorrect.

b. The personal evaluation of personal wishes, though personal, is also correct or else it is incorrect.

c. Man can be denied the fulfillment of a personal wish but he cannot be denied the fulfillment of his personal evaluation. This occurs as the evaluation is made. With understanding our will becomes free to choose the correct evaluation and the constructive way of dealing with the nervous crisis.

5. A case report illustrating the resolution of a nervous crisis is included.

CONNECTIVE TISSUE CHANGES IN PULMONARY DISEASES

Martin FitzPatrick, M.D., Kansas City*

In recent years some biochemical studies have been conducted on the fibrous elements of connective tissue of several organ systems. Our studies have dealt with the elastic fibers of lung and aorta of various species, and in the normal human subject. More recently studies have been conducted to define the amino acid composition of pulmonary elastin, both in the normal human subject, and in the presence of various diseases.

The mode of extraction of elastic fibers from pulmonary connective tissue can cause important changes in amino acid composition. It has been shown that alkaline digestion reduces the amount of serine, threonine, methionine, and arginine. Histidine, aspartic acid and glutamic acid are also found in reduced quantities in elastic fibers isolated by alkaline digestion.

Preliminary studies disclose no significant alteration in amino acid composition of pulmonary elastin in patients with tuberculosis. Pulmonary emphysema, an important degenerative disease of the lung characterized by loss of elasticity, has been recently considered to be a connective tissue disease. We have found no striking difference in amino acid composition of pulmonary elastin from patients with pulmonary emphysema. In male patients with bronchogenic carcinoma, an abnormal collagen appears to be present, in that it resists complete digestion by the enzyme collagenase. This has not been observed in other diseases or in the normal individual. Further studies concerning the origin and significance of this abnormal collagen are in progress.

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VANCOMYCIN IN STAPHYLOCOCCAL BACTEREMIA

Robert Weber, M.D., Kansas City*

From 1957 through 1960, hemolytic coagulase positive staphylococci were isolated from quantitative blood cultures in thirty-six patients at the University of Kansas Medical Center. Two or more cultures were positive in thirty-three of these patients with staphylococcal bacteremia. It was observed that the first culture was most likely to be positive if a bacteremia was demonstrated and that all subsequent cultures were also positive until treatment was instituted. Seventeen of these patients were treated with Vancomycin therapy. Eleven of these patients had endocarditis, three had osteomyelitis, two had pneumonia and one had a cavernous sinus thrombosis associated with a septicemia. Seven patients with staphylococcal bacterial endocarditis were successfully treated with Vancomycin therapy. Two patients who had a plastic prosthesis in the heart, following open heart surgery, were treated with Vancomycin and other antibiotic therapy unsuccessfully. One patient with staphylococcal endocarditis had a bacteriologic cure with Vancomycin therapy, but died as result of a ruptured aortic valve. The Vancomycin therapy was effective in treating staphylococcal septicemia when the organism was resistant to penicillin. Thrombophlebitis and chills and fever were common side effects of Vancomycin, but were not severe. The incidence of the side effects was reduced by administering the Vancomycin intravenously in five to ten minutes by syringe. Thrombophlebitis occurred in all patients who received the Vancomycin by continuous intravenous drip. The usual amount of Vancomycin administered was 2 grams daily for 14 to 21 days.

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VISUAL LOSS IN THE OLDER PATIENT: TEMPORAL ARTERITIS

Joseph M. Stein, M.D., Topeka

Two cases of temporal arteritis with amaurosis, bilateral in one and unilateral in the other, were presented. Both of these patients represented diagnostic problems for one or more months before the diagnosis was established and treatment was given. The early symptoms may be only headache and variable visual disturbances. Treatment is symptomatic, with the greatest benefits being obtained from the judicious use of the corticosteroids. The current literature was briefly reviewed. Emphasis was placed on the importance of the preservation of vision in the elderly person to try to prevent a catastrophic disability in persons who otherwise might be quite self-sufficient. Early recognition of the sometimes puzzling syndrome was stressed for this reason.

[This paper is to be published in full in a later issue of the JOURNAL—Ed.]

THE CLINICAL PICTURE OF SARCOIDOSIS: A REPORT OF EIGHT PROVED CASES

*Joseph N. Plumer, M.D. and Albert Jackson, M.D., Wadsworth**

The diagnosis of sarcoidosis is still difficult and only awareness will lead one to the correct diagnosis. X-ray of the chest is usually the first clue to awareness of this condition. The diagnosis can be definitely established by biopsy of a lymph node or other organ.

The Kveim reaction, which has many false negatives, is not reliable. It is difficult or impossible to obtain, when needed.

Bone involvement is a late manifestation of the disease, occurring in approximately 15 per cent to 25 per cent of the cases.

Elevated serum calcium is found when there is osteoporosis, which is not always present, and a normal serum calcium does not rule out sarcoidosis. A/C ratio changes and an altered electrophoretic pattern are not always present in the early stages of the disease and, therefore, non-specific.

It is our opinion that sarcoidosis is a hypersensitivity disease.

A differentiation between sarcoid type reaction and systemic sarcoidosis is only quantitative and not justified.

Steroids are the only effective treatment in sarcoidosis and all patients should be treated as soon as diagnosed.

The prognosis is not always benign. Sudden death does occur.

* Wadsworth VA Hospital, Wadsworth, Kansas.

Editor's Note: This paper will be published in THE JOURNAL in its entirety in a subsequent issue.

EOSINOPHILIC GRANULOMA OF THE SKULL: REPORT OF A CASE

W. Graham Calkins, M.D., and Charles E. Brackett, Jr., M.D., Manhattan

A case is reported of a young woman with a single lesion of eosinophilic granuloma of the skull. The lesion invaded the temporal muscle and the dura producing convulsive seizures secondary to brain compression. This case is unique in that it is the only instance in which a convulsive disorder was produced by eosinophilic granuloma. Surgical excision of the lesion was curative. The signs, symptoms, pathology and treatment of eosinophilic granuloma were discussed. The interrelationship between eosinophilic granuloma, Letterer-Siwe disease and Hand-Schuller-Christian disease were elaborated upon, and soft tissue lesions of eosinophilic granuloma were discussed.

FOLIC ACID AND METABOLISM OF THE LIVER DISEASE

*Robert T. Manning, M.D., Kansas City**

The appearance of macrocytic anemia in individuals with cirrhosis has been attributed to folic acid deficiency. Such deficiency might conceivably be due to decreased intake, decreased absorption, altered conversion to the active biological folic acid coenzyme, increased inactivation or other causes. In an attempt to assay folic acid deficiency in patients with cirrhosis, the enzymatic determination of formominoglutamic acid in the urine before and after histidine loading was attempted. Formominoglutamic acid is a natural metabolite of histidine the degradation of which requires the presence of sufficient quantities of folic acid to act as coenzyme; therefore, deficiencies of folic acid at the functional site may appear indirectly as excretion of increased quantities of formominoglutamic acid in the urine following histidine loading.

Preliminary studies utilizing this technique of a loading dose of two grams of histidine have indicated that patients with macrocytic anemias and liver disease may be folic acid deficient. Four of six such patients showed an increased excretion of formominoglutamic acid over their control value and over comparable normal values. Interestingly, one patient with myocarditis showed the most marked elevation of formominoglutamic acid excretion of any patient so far studied.

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ELECTROCARDIOGRAPHIC STUDIES IN CONGENITAL HEART DISEASE

*James E. Crockett, M.D.,** *and William Hayes, M.D., Kansas City*

A detailed analysis has been made of the electrocardiographic findings in patients with congenital heart disease in whom the catheterization diagnoses have been proven by surgery or autopsy.

Right axis deviation and right ventricular hypertrophy were common findings in cyanotic forms of congenital heart disease and were seen typically in certain acyanotic forms such as pulmonary stenosis. The degree of right ventricular hypertrophy and the QRS-T changes were found to vary widely, however. Systolic overloading of the right ventricle was found in a variety of conditions, i.e. pure pulmonary stenosis, tetralogy of Fallot, trilogy of Fallot and transposition of the great vessels, but there were important changes between these groups. In tetralogy of Fallot, for example, in which the ventricular septal defect allows equalization of right and left ventricular pressures, the T wave is rarely inverted beyond V1. In pulmonary stenosis or trilogy of Fallot with intact ventricular septum, the right ventricular pressure often becomes quite high and T wave inversion through V3 is common. This is frequently accompanied by a qR complex in V4R and V1 as contrasted to the slurred upstroke of the R wave in V4R and V1 usually seen in tetralogy of Fallot.

In transposition of the great vessels the right ventricular hypertrophy is quite common and expected, but when left ventricular hypertrophy accompanies, the compensating lesion will be ductus arteriosus or ventricular septal defect.

The ostium secundum type of atrial septal defect is reflected by right axis deviation and right bundle branch block (diastolic or volume overloading of the right ventricle). However, right bundle branch block and left axis deviation of 0 to minus 60 degrees usually indicates an incomplete atrioventricular canal or ostium primum defect while a marked left axis deviation (minus 60 to minus 120 degrees) and right bundle branch block points to a diagnosis of complete AV canal.

Tricuspid atresia was manifested by evidence of left ventricular hypertrophy or an unusual degree of left axis deviation for the age.

Ventricular septal defect in infants and small children was usually accompanied by

biventricular hypertrophy. In older patients, diastolic overloading of the left ventricle was the common finding unless increasing pulmonary vascular resistance led to systolic overloading of the right ventricle. In the usual case of septal defect or ductus arteriosus, the development of pulmonary hypertension eventually produced evidence of systolic overloading of the right ventricle.

Coarctation of the aorta and congenital aortic stenosis resulted in varying degrees of left ventricular hypertrophy, usually of the systolic overloading type.

Truncus arteriosus did not reveal any diagnostic EKG findings.

The usual EKG changes in Ebstein's malformation are large P waves, first degree heart block and right bundle branch block.

Many other detailed observations have been made. These studies have shown that a careful analysis of the EKG in congenital heart disease will produce accurate information regarding the pathologic physiology of the lesion and will permit an accurate diagnosis in many cases.

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IODIDE GOITER

David Lukens, M.D., Hutchinson

This report concerns a 36-year-old woman with a lifelong history of asthma who has been taking medication from a Biloxi, Mississippi, clinic, which medication included among other things Potassium Iodide and Fowler's solution. She presented herself complaining of severe constipation and lassitude with easy fatigability. The physical examination revealed signs of advanced hypothyroidism with a thyroid gland symmetrically enlarged about 3 times normal size. BMR was +3. Radioactive iodine uptake study was in the myxedema range. The differential diagnosis included Hashimoto's disease, iodide goiter, and arsenic intoxication. Biopsy of the thyroid revealed a hyperplastic gland. Chemical analysis of the finger and toe nails revealed minute concentration of arsenic. Following discontinuation of asthma medication the patient improved considerably but leveled off still in a hypothyroid range, and treatment with Tri-iodiothyronine brought the patient into a euthyroid state, whereupon the gland returned to normal size. Of principal interest in this case is the variation of the physiological response to radioactive iodine, because of the fact that her radioactive iodine uptake remained in the myxedema range following discontinuation of Potassium Iodide ingestion. The aspects of this physiological variation will be discussed.

Editor's Note: This paper will be published in THE JOURNAL in its entirety in a subsequent issue.

PAROXYSMAL NOCTURNAL HEMOGLOBINURIA: SOME CLINICAL OBSERVATIONS

Newman V. Treger, M.D., Topeka

The immunology of hematology has been of considerable interest in recent years, with the breakthroughs provided by Dr. Carl V. Moore, his associates, and others. Paroxysmal nocturnal hemoglobinuria is a rare disease which, with its hemolytic anemia, provides an opportunity for study of some of the lesser understood mechanisms. Here, the chief defect is thought to be in the erythrocyte. In fact, a positive diagnosis cannot be made, except by the demonstration of the characteristic erythrocyte-abnormality. Several hundred cases have been diagnosed and reported in the world's literature. Several therapies have been tried during the seven and a half years of clinical observation in a young matron with paroxysmal nocturnal hemoglobinuria. These observations will be reported, along with summarization of a review of the world's literature.

TECHNIQUES OF INTRAMUSCULAR INJECTION. THE AVOIDANCE OF NEEDLESS PAIN AND MORBIDITY

Samuel Zelman, M.D., Topeka*

The traditional technique of posterior gluteal intramuscular injection was reviewed, emphasizing the necessity for meticulous care in details if needless pain and morbidity are to be avoided. A questionnaire of nursing practice disclosed remarkable deviation from accurate technique, and revealed a high incidence of needless infliction of pain. Case examples of sciatic neuritis were omitted because of time limitation.

The new Swiss technique utilizing ventral gluteal musculature is free from hazard of injury to large vessels or nerve trunks. In two years' experience, it proved free of morbidity, simple to teach and practice, and almost foolproof of error. It deserves wide adoption.

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[This paper will be published in full in an early issue of *The American Journal of the Medical Sciences*.—Ed.]

CLINICOPATHOLOGICAL CONFERENCE

This was the first KUMC admission for this 68-year-old farmer.

Chief Complaint: Severe pain in the right inguinal region of three weeks duration.

History of Present Illness: The patient was in his usual state of health until three weeks before admission when he developed severe pain in his right inguinal region. The pain radiated into the right testicle, the inner aspect of the right thigh, into the back and the left abdominal region. Associated with the pain was frequency of urination, dysuria, anorexia and fever, but no nausea, vomiting or diarrhea. For two weeks before admission he had noted bright red blood in his stools. For many years he had had a recurrent low abdominal pain associated with constipation and relieved by laxatives. He was afforded some relief from the pain by assuming a sitting position with his legs flexed and drawn toward his chest. About two weeks before admission he had a sudden onset of numbness and weakness of his right arm and leg associated with some mental confusion. One week before admission he was hospitalized elsewhere. He continued to have pain, and he was subsequently sent here for evaluation.

Past History: He had had the usual childhood diseases. Two years before admission here he was hospitalized elsewhere for arthritis. For the past two years he had been taking digitalis which had originally been given to him because of dependent edema. He smoked two packs of cigarettes daily for 54 years, and he had consumed large amounts of alcohol.

Family History: His mother died at age 69 of diabetes mellitus; his father died at age 72 of cancer of the liver. One brother had peptic ulcers; one brother had asthma.

System Review: During the previous winter months he had had severe headaches, dyspnea on exertion, a chronic cough productive of sputum, 3 pillow orthopnea, paroxysmal nocturnal dyspnea and progressive pedal edema. He had also had an unknown amount of weight loss.

Physical Examination: The patient was a well developed, well nourished, white man who was confused and agitated and in acute distress, sitting doubled up in bed and complaining of right inguinal pain. His pulse was 65 and regular; blood pressure, 115/65; temperature, 99°; respiration, 20. His skin and mucous membranes were dry. His tongue was coated, and the pharynx was red. The chest was hyporesonant to percussion, and there was an increase in the anterio-posterior diameter. Rales which cleared on coughing were heard in both bases. There was a presystolic gallop rhythm. The abdomen was soft but somewhat tender in the right mid and lower quadrants. The liver was palpated two fingerbreadths below the costal margin and was somewhat tender. Bowel sounds were active. No unusual masses were palpated. There was 2 plus pitting edema of the lower

legs. Rectal examination was negative except for diffuse tenderness. There was questionable left facial weakness and questionable deviation of the tongue to the right.

Laboratory Data: The specific gravity of the urine was 1.025 with trace of albumin, 1-2 hyalin cast, 2-4 pus cells/hpf, reaction PH 5. The hemoglobin was 13.5 gm.; hematocrit, 44. A white count on admission was 18,600 with 73 per cent polys (70 per cent filamented, 3 per cent non-filamented), 20 per cent lymphs; 5 per cent eosinophiles, 2 per cent monocytes. On the third hospital day a white count was 15,830 with 90 per cent polys (69 per cent filamented, 21 non-filamented), 9 per cent lymphocytes, 1 per cent basophiles. A sedimentation rate was 24 mm. in 30 minutes, 25 mm. in 60 minutes. The serology was non-reactive. BUN on admission was 34 mg. per cent; one day before death, 132 mg. per cent. Blood glucose was 95 mg. per cent; sodium, 132 to 141 mEq; potassium, 5.0 on admission, 5.2 on the day before death; chlorides, 94 to 99 mEq. Total proteins were 6.4 gm. per cent; serum albumin, 2.73, globulin, 3.67; lipase, 0.9 units; lucine aminopeptidase, 155; SGOT, 35; SGPT, 18; calcium, 4.9; phosphorous, 1.9. Cholesterol was 120 (59 per cent esters). Bilirubin total was 0.2 (direct 0.1); alkaline phosphatase, 3.7; acid phosphatase, 0.4; thymol turbidity, 11; prothrombin time, 75 per cent; cephalin flocculation, 2. Skin tests for histoplasmin and intermittent Tbc were negative at 48 hrs. A urine culture was negative. Spinal fluid showed protein 30 mg. per cent, 1 Rbc, collodial gold 0011100000.

Hospital Course: The patient continued to complain of right inguinal pain and maintained a position with his legs flexed and drawn toward his chest. He was placed on a low sodium diet and given digitalis and codeine. On the second hospital day multiple successive generalized convulsions developed but were effectively treated with sedatives and CO_2 inhalations. A lumbar puncture showed an opening pressure of 130 mm. of clear spinal fluid. Following the seizures his temperature was 104.2°. He was given diphenylhydantoin sodium and barbiturates. The abdominal pain continued. On the fourth day he developed abdominal distention and urinary retention necessitating the insertion of a Levine tube and Foley catheter. The next day an exploratory laparotomy was performed after which he ran a septic course with rectal temp. of 102-103°. He was restless and confused. On the second post-operative day he became hypotensive and respiration was labored. Despite antibiotics, intravenous fluids and whole blood transfusions, he became comatose and developed Cheyne-Stokes respiration, and he died at 5:07 p.m. on the seventh hospital day.

Operative Findings: Peri-appendiceal abscess and localized pelvic peritonitis.

Final Diagnosis: Peri-appendiceal abscess and local pelvic peritonitis.

(Editor's Note: This is the first occasion of THE JOURNAL publishing abstracts of the papers presented at a scientific meeting other than meeting of the Kansas Medical Society. It was made possible on this occasion by the efforts of Dr. D. R. Bedford, of Topeka, who was the chairman of the program committee for the meeting. We appreciate his efforts to obtain these abstracts, and hope that they will prove to be useful to our readers.

The Editorial Board would be pleased to obtain similar abstracts of papers presented at other meetings in the state, if those concerned with the meeting would tell us about them.)

Ammonia and Leukemia

Observations on Blood Ammonia in Leukemic Patients

WILLIAM E. LARSEN, M.D.,** ***Kansas City***
ROBERT T. MANNING, M.D.* ***and***

DISPUTE EXISTS REGARDING the source of the ammonia measurable in blood. That generation of ammonia following shedding of blood may occur has been amply documented, but it has been confirmed that following collection of blood samples the *in vitro* generation of ammonia is negligible for approximately twenty minutes.[1, 2] We have had occasion to observe four patients in which *in vitro* (and possibly *in vivo*) production of ammonia was markedly altered, producing high measured levels of blood ammonia which did not reflect the true ammonia content of the patients' blood. Two of these patients had chronic myelogenous leukemia and one had chronic lymphatic leukemia. One presented, in addition, rather marked asymptomatic "hypoglycemia."

Case No. 1. K.M., KUMC No. 53-11366.

This 48-year-old white woman was admitted for the third time to the University of Kansas Medical Center on November 3, 1958 with the established diagnosis of chronic myelogenous leukemia. Four weeks prior to admission she had noted the onset of exertional dyspnea and orthopnea. These symptoms gradually increased in severity and were associated with the appearance of pedal edema and swelling of her abdomen. She gained 26 pounds in the five months prior to admission. No fever or chills had been present.

Physical examination revealed a dyspneic, emaciated woman with pitting edema and marked ascites accumulation; blood pressure 104/70; respirations 30. Moist rales and decreased breath sounds were present at both lung bases, and a grade II systolic murmur was present over the precordium. The liver was palpable 6 fingerbreadths below the right costal margin. The spleen was surgically absent.

On November 4, 1958 a blood sugar was reported as 5 mg. per cent, at which time the patient had no symptoms of hypoglycemia. This was repeated on November 5, 1958 with sodium fluoride as the anticoagulant and reported as 58 mg. per cent. On November 13, 1958 the blood ammonia was noted to be 310 mcg. per cent (N-70-130), and on November 14, 1958 it was 1,100 mcg. per cent. An electroencephalogram on November 13, 1958 was normal. No manifestations of ammonia intoxication were apparent.

The studies detailed below were carried out on November 18, 1958, at which time her total Wbc. was 192,000 with 73 polys (12 nonfilamented), 2 lymphocytes, 6 eosinophils, 4 basophils, 3 metamyelocytes and 12 myelocytes. Platelets were 78,000; hemoglobin 10 Gm. per cent; bromosulphthalein 11 per cent in 45 minutes. SGOT was 10; total serum bilirubin 0.2, direct 0.1; alkaline phosphatase 4.7 mM. units; cepalin cholesterol flocculation negative; thymol turbidity 2 units; serum albumin 2.82 Gm. per cent, globulin 2.89 Gm. per cent; total cholesterol 196 mg. per cent, esters 56 per cent.

Three patients with leukemia (two with chronic myelogenous, one with chronic lymphatic) are reported who showed high leukocyte counts, high blood ammonias and low blood sugars on routine laboratory examination. Studies are reported showing a rapid increase in ammonia content and lowering of glucose in the blood on standing for brief periods of time. This phenomenon is believed to be due to the metabolic activity of the increased white cell mass. Its significance is discussed.

Medication included chlorothiazide (1 Gm. daily) from November 5, 1958 to November 11, 1958, and 6-mercaptopurine (150 mg. daily) from November 12, 1958 to November 19, 1958, and 100 mg. on November 20, 1958 to November 21, 1958.

She was discharged on November 21, 1958 (moderately improved), and with a weight loss of 19 pounds.

Case No. 2. E.W., KUMC No. 58-2840.

This 79-year-old white woman was first hospitalized in February, 1958, at which time a carcinoma of the hepatic flexure was surgically removed. No metastases were found.

She did well until three months prior to admis-

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sion, when she noted the onset of easy fatigue and tiredness. These symptoms increased in severity, and she developed anorexia and nausea but no vomiting or diarrhea.

She was admitted to the University of Kansas Medical Center for the second time on June 27, 1960, at which time examination revealed a frail woman who appeared chronically ill. Blood pressure was 140/60; pulse, 78. Grade II arteriosclerotic retinopathy was present. A few moist rales were noted in both bases and a grade II systolic ejection murmur was audible. Abdominal examination revealed the liver palpable 3 fingerbreadths below the right costal margin and the spleen was palpable 4 fingerbreadths below the left costal margin. No other gross abnormalities were noted.

The initial blood count revealed 337,000 Wbc. with 43 polymorphonuclears (21 non-filamented), 1 eosinophil, 1 basophil, 24 metamyelocytes, 28 myelocytes and 3 blast forms. Platelets were 464,000; hemoglobin, 7.7 Gm. Total serum bilirubin was 0.4, direct, 0.1; alkaline phosphatase, 1.3 mM. units; cephalin cholesterol flocculation negative; thymol turbidity, 5; serum albumin 3.30; globulin 7.01; total cholesterol, 129; ester, 59 per cent. BMR was +27 per cent. Bone marrow examination showed findings diagnostic of chronic myelogenous leukemia.

On July 5, 1960 a blood ammonia was 386 mcg./100 ml.; no symptoms or signs of ammonia intoxication were present. The studies reported below were done on July 7, 1960. She was started on therapy on July 7, 1960 and was discharged on July 9, 1960.

This was the first admission for this 45-year-old man, who complained of tiredness and weakness of three weeks' duration. He had had a 30-pound weight loss in the preceding four months, and had had frequent upper respiratory infections for three years before admission.

The patient was a well-developed, well-nourished man who did not appear ill. His blood pressure was 150/95. There were palpable cervical nodes, mucosal pallor and a thyroglossal cyst. Chest and cardiac examination revealed a grade I basal systolic murmur. The liver was felt 3 fingerbreadths below the right costal margin; the spleen was enlarged to the iliac crest.

The admission laboratory examination showed a Wbc. of 416,000, 95 per cent lymphocytes; platelets were 114,000; hemoglobin, 7.4 Gm. Bromsulphthalein retention was 10 per cent. Total serum bilirubin was 0.5, direct 0.1. Alkaline phosphatase was 1.9 mM. units; total protein 7.10; serum albumin 4.02; globulin 3.08; cephalin cholesterol flocculation 14; thymol turbidity 12 units. Total cholesterol was 135 mg. per cent, esters 70 per cent. Bone marrow revealed findings diagnostic of chronic lymphatic leukemia.

The laboratory findings noted in Figure 1 were done on the fourth hospital day. The patient was not receiving any medication.

Methods

Blood was obtained from each patient and an aliquot immediately placed in cold trichloroacetic acid and sodium fluoride to stop enzymatic action and glycolysis. The remainder of the sample was placed in tubes with sodium citrate (Cases 2 and 3) or heparin (Case 1) as anticoagulant, and aliquots taken at intervals for determination of ammonia and glucose.

Ammonia was determined by Conway's method, using the TCA filtrates. Glucose was determined in Case 1 by Somogyi's method and in Cases 2 and 3 by the use of "Glucostat."[3]

Results

The ammonia and glucose values are shown in *Figure 1.*

Discussion

The clinical picture of "low blood glucose," "high blood ammonia" and absent associated symptomatology suggests that the chemical alterations measured result from the *in vitro* metabolic activity of the large circulating leukocyte mass. An elevated basal metabolic rate and occasional observation of low blood glucose in leukemic patients has been previously noted. These have been attributed to utilization of the glucose by the leukemic cells and the increased oxygen consumption of this large abnormal cell mass. Studies related to carbohydrate metabolism

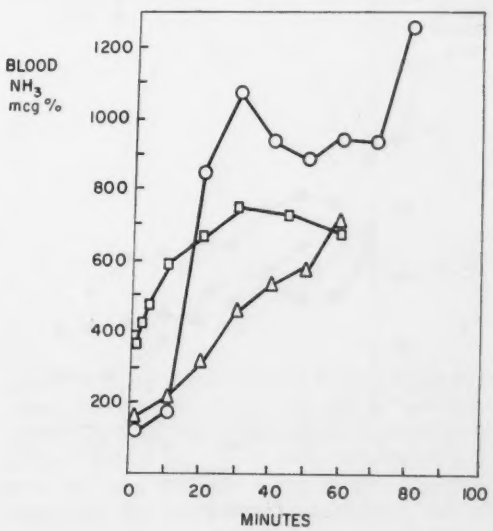

Figure 1

have indicated that the leukemic cell differs from its normal counterpart in enzyme content and enzyme activity.[4, 5, 6]

Little is known relative to the nitrogen and amino acid metabolism of these cells. Waisman[7] and Rouser[8] have noted that glutamic dehydrogenase is increased in leukemia cells and that granulocytic leukemia cells contain no detectable glutamine. Rouser also reports that the plasma glutamine content of patients with chronic granulocytic leukemia (with predominantly polymorphonuclears) is reduced. Since glutamic acid can support cell metabolism in part by entering the Krebs cycle as α-ketoglutarate following deamination of transamination and glutamine as a reversible "store" of ammonia, it seems feasible that the ammonia noted in these sera may be due to active deamination of glutamine and glutamic acid. This process conceivably could be stimulated by decreasing availability of glucose as the plasma glucose is utilized on standing (i.e., gluconeogenesis).

Other possibilities as a source of the ammonia formed are: 1) deamination of other amino acids, 2) proteolysis with ammonia liberation, or 3) deamination of other nitrogenous materials (i.e., adenine by adenylic deaminase).[2]

The occurrence of this "laboratory phenomenon" in patients with leukemia is worthy of note, since it can cause clinical confusion if one is not aware of its occurrence, and it pointed out to us the paucity of information regarding the amino acid and nitrogen metabolism of the normal and abnormal leucocyte.

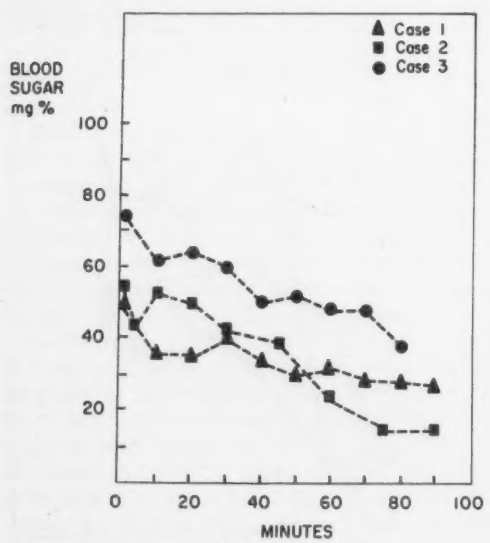

Figure 2

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Editor's note: This article was submitted to The JOURNAL for publication in March, which is the University of Kansas Medical Center issue. Due to space limitations at that time, The JOURNAL was able to publish only half of the articles submitted.

The remaining papers will appear in the upcoming issues.

The JOURNAL would like to thank these authors for being so patient while waiting for their article's publication.

Childbearing is more hazardous for teen-agers than for women in their early twenties, according to statistics cited by *Patterns of Disease*, a monthly Parke, Davis & Company publication for physicians. Mortality rate of pregnant teen-agers is 2.5 for every 10,000 live births; among those in their early twenties the rate is only 1.9 for the same number of live births.

Greatest menace to the health of pregnant women is eclampsia, a form of toxemia, which in their first pregnancy, tends to attack teen-agers 15 times more often than older women. Among pregnant women who had been pregnant before, the teen-ager is four times as prone to eclampsia as older women.

In a study of 1,500 teen-age maternity patients, a correlation was found between weight gains of 40 pounds or more and the incidence of eclampsia, although *Patterns* points to another study which indicates that overweight did not appear to be a direct cause of eclampsia.

The pipe draws wisdom from the lips of the philosopher, and shuts up the months of the foolish; it generates a style of conversation contemplative, thoughtful, benevolent and unaffected.

—*William Makepeace Thackeray*

Torticollis

The Treatment of a Case of Spasmodic Torticollis by Means of Hypnosis

WILLIAM SEEMAN, Ph.D.,* ***Kansas City***

THIS PAPER CONSTITUTES a brief clinical report on the treatment by hypnotic technique of a young man with spasmodic torticollis. The patient was referred to the Department of Psychiatry by a neurosurgeon, and the treatment was undertaken early in January, 1960.

The patient was married, had one child, and was employed in a task which, in effect, isolated him from any other people. He spoke of this as being "practically pensioned" by the company; and, in effect, he spent a great deal of his time in reading. The position of his head and neck is indicated in *Fig. 1*. Understandably, he had a great deal of difficulty not

Figure 1

only driving his car, but executing a large number of other normal functions. Socially, he might be characterized as living the life of a recluse. On his first visit and on a number of other visits he wore around his neck a rubber support which he subsequently discarded.

The patient reported that his condition had been "bad for about 27 months. My neck bothered me some—a little, since I was fifteen. It just didn't *feel* right. It didn't feel like it worked right." He recalled that at age 15, while playing football "I really banged down hard on my head." At that time, too, he felt "frustrated; I wasn't making much headway. I was on the B-squad with the scrubs. It worried me more than anything in my life! It started happening then."

The patient reported that he had seen a number of different physicians who had given him a variety of diagnostic impressions. The diagnoses mentioned by the patient were "cerebral palsy, encephalitis, Parkinsonism, torticollis, and nerves." He reported that the referring physician felt that "it could be psychogenic or physical."

A personality test known as the Minnesota Multiphasic Personality Inventory (MMPI) was administered. This test yields a set of scores on a variety of psychological or psychiatric scales, and these scores can be plotted as a personality profile. The MMPI personality profile for this particular patient is presented in *Fig. 2*. Scores between 30 and 70 (see T-scores along the left hand margin) are regarded as being within "normal limits." Scores at or above the 70 magnitude are regarded as "abnormal" scores. The particular profile pattern observed in this patient's MMPI (*Fig. 2*) is most revealing; for, not only are there two scale scores in the "abnormal" range, but the *particular* scales elevated and the

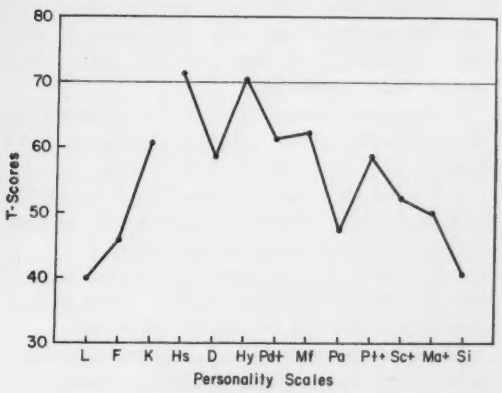

Figure 2

* Department of Psychiatry, University of Kansas School of Medicine.

shape of the profile are of great significance. The elevated scales 1 and 3 (Hs and Hy) are *somatic* scales; and indicate a personality and an individual who is likely to express his emotional difficulties and his psychological conflicts through bodily symptomatology (in contrast, for instance, to one who is likely to express them, say, in suspecting the intentions of other people or in aggressive personal relationships). Moreover, the first three scales of the profile will be seen to form a "V" shape. This, in the literature of the MMPI, has become known as the "conversion V." In other words, an individual with *this kind* of MMPI profile pattern is likely to present itself as *physically ill* with symptoms which, however, should be suspected as being of "psychogenic" origin; or as having a "psychogenic component." At any rate, it was this MMPI personality profile pattern which suggested a "psychogenic" or psychological basis for the disorder, and which suggested that hypnosis might conceivably be helpful in dealing with the spasmodic torticollis.

There is no one single and approved way of using hypnosis in a case of this sort. One possibility would have been to combine psychotherapy with hypnosis and to use a "hypnotherapeutic" technique. This would involve first hypnotizing the individual and then "exploring" and perhaps "uncovering" significant factors in the life history of this patient connected with the torticollis. An alternative method, and the one decided on here, involves no "uncovering" techniques, but relies, rather, on posthypnotic suggestions. This latter technique is sometimes characterized as "symptomatic" treatment, and it must be admitted that there are therapists who tend somewhat to frown on such techniques. At least three arguments have been adduced against such "symptomatic" treatment:

1. Since such treatment does not eliminate the "dynamic cause" of the symptoms, remission of the symptom must at best be of short duration.

2. For the same reason, remission of a symptom must inevitably be followed by a substitute symptom (sometimes "worse" than the original).

3. Remission of symptoms with this kind of treatment has on occasion been followed by exceedingly disturbing episodes in the life of the patient.

Whatever may be said for or against these arguments, it can be said with absolute confidence that there is no experimental evidence available to support a cause-and-effect relationship relevant to any of the three arguments. Clinical evidence *against* the *inevitability* of 1 and 2 above is certainly available. An unpublished survey by Roy Dorcus, read at the hypnosis symposia in Lawrence in 1960, provides good evidence on this. With respect to number 3, the evidence is fragmentary, highly anecdotal, and absolutely unquantified.

Hypnotic sessions were arranged on a once-a-week basis with the patient. The objective in the first few sessions was simply to achieve "depth" in order to make the suggestions subsequently effective. In the later sessions such posthypnotic suggestion was given while the patient was under hypnosis. These suggestions all centered around the notion of "muscle relaxation"; i.e., the suggestion was given that the neck muscles would gradually relax, that the "natural and normal" use of the neck and head would be recovered. On the sixth visit the patient purported to experience some change, but objective evidence of this was not obvious; the improvement claim was looked on dubiously, though the patient was, of course, never told that this was the case. In subsequent visits, however, it became evident that marked changes were occurring. The frequency of these hypnotic sessions was reduced, and the patient continued to improve. By June it would probably have been impossible for an observer to detect any difficulty. At that point the patient began to return to a more adequate social life, found himself able comfortably to visit friends, etc. Some notion of his condition at that time can be obtained from *Fig. 3.*

Figure 3

Subsequent follow-up periodically has indicated that the symptom of spasmodic torticollis remains in full remission. The patient has now demonstrated a willingness to explore the possibilities of more adequate and more remunerative vocational objectives. His latest communication registered the information

(Continued on page 317)

Lymphadenopathy, Hepatosplenomegaly, Anemia and Skin Lesions

CASE PRESENTATION

A 13-MONTH-OLD WHITE GIRL was admitted to KUMC for the first time on October 28, 1960, with swelling of the neck, groin and axilla, and pallor and weakness of two months' duration. In May, 1960, clear vesicles developed on the genital labia and became purulent and associated with erythema. On August 18 inguinal swelling developed, and within two weeks she had cervical swelling, fever, pallor and weakness.

The child was born in Oklahoma on September 23, 1959. This was the second pregnancy for the mother, and delivery was at full term, normal, and without complications. The baby's birth weight was 8½ pounds. She had been breast fed, and at the time of her hospitalization she was eating table foods including meat, eggs and vegetables. She sat up at 5½ months of age, and she began to walk at 12 months of age. She had had three DPT and two polio injections, but no smallpox vaccination. She had had no other illnesses or any known allergies. She had had some anorexia during the week before admission.

The baby's father was 34 years old, and her mother was 30 years old. Both were in good health. She had one sister, six years old, who was living and well. A maternal grandfather had diabetes.

The patient was a well developed, pale, white baby girl. Her rectal temperature was 99.2 degrees F.; pulse rate, 160 per minute; respirations, 40 per minute; weight, 23 pounds. Her head was normal. There was no inflammation or exudate from the ears, nose or throat. There were 10 x 10 cm. matted, firm, tender, bilateral lymph nodes in the neck, and nodes ranging from 2 to 5 cm. in diameter in the axilla. The chest was clear and resonant to auscultation and percussion. The heart was of normal size; the rhythm was regular, and no murmurs were heard. The liver was palpable 2 cm. below the right costal margin; the spleen was palpable 1 to 2 cm. below the left costal margin. There was a bluish discoloration of the umbilicus. There was marked inguinal lymphadenopathy with biopsy scars bilaterally. There was a petechial rash over the inguinal region, abdomen, thighs and scalp. The neurological examination was negative.

On admission the specific gravity of the urine was 1.034 with a pH of 5.5, 1 plus albumin, negative sugar, and no abnormal microscopic findings. The leukocyte count was 10,500 with 59 per cent polymorphonuclear neutrophils (56 per cent filamented, 3 per cent non-filamented), 35 per cent lymphocytes, 2 per cent basophils, 4 per cent monocytes, and 1 nucleated red blood cell. The hemoglobin was 7.7 gm. per cent with a hematocrit of 24. On October 29 platelets were 80,000. On November 10 the serum albumin was 2.41 gm. per cent; serum globulin, 1.37; total protein, 3.78. Tuberculin (PPD), histoplasmin and Frei skin tests were negative.

The patient had fever with spikes to 104 degrees. The hemoglobin remained low despite transfusions. Her appetite was poor, and she was irritable. The petechial rash became more dense. On November 5 a caseous exudate developed on her tonsils, bilaterally. A culture grew out pneumococcus and Neisseria. On November 8 a purulent discharge developed from the right tympanic membrane. On November 14 a portion of the left tonsil sloughed off. A culture of the sloughed area grew E. coli. On November 17 the left tympanic membrane began to drain. From November 10 to November 21 she had loose and occasionally watery diarrhea. On November 11 corticosteroid therapy was started with remission of the fever and definite regression in the size of the lymph nodes. On November 23 some dependent edema developed. On November 28 and 29 she had a five pound weight loss, and the edema disappeared. Her anemia was stabilized; the hemoglobin increased to

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9.3 gm. on the day of discharge. She continued to have hepatosplenomegaly and a petechial rash. On November 30 she was discharged and followed as an outpatient.

While at home her appetite improved, and she seemed to do well. On December 9, however, a papular rash developed over her arms and scalp, and within four days it had become vesicular and progressed to involve her trunk. On December 15 she had a fever of 101 degrees. She took fluids poorly.

She was admitted here for the second time on December 16, 1960, with a generalized papulovesicular rash on her palms, the soles of her feet, trunk, extremities and scalp as well as vesicles on the mucous membranes of her mouth.

At that time the leukocyte count was 30,250 with 60 per cent polymorphonuclear neutrophils (32 per cent filamented, 28 per cent non-filamented), 18 per cent lymphocytes, 2 per cent basophils, and 28 per cent monocytes. The hematocrit was 37.

On the morning of December 18 her breathing became more labored, and by late afternoon the respiratory rate was 60 to 70 per minute. Peripheral pulses were 150. Her color was pale, and her extremities were cool. The chest was clear. Within three hours the respiratory rate climbed to 80 per minute, and she had audible rhonchi and rales. She became increasingly more dyspneic and less responsive, and she died quietly at 11:45 p.m.

Dr. Mahlon Delp (moderator): Are there any questions?

Maurice Cashman (Fourth year student):* Had she been exposed to any infectious diseases, exanthemata, fungus diseases, or tuberculosis?

Dr. Russell Etzenhouser (resident in pediatrics): On her previous admission in November there were several cases of exanthemata on the same floor, but she had not previously been exposed before her hospitalization.

Mr. Cashman: Had she recently been vaccinated for smallpox?

Dr. Etzenhouser: There is no record of it.

Gordon Ewy (student): Will you describe the skin lesions in more detail? Did they ever go away? Did she have any vesicles? Was there any purpura in the external auditory canal?

Dr. Etzenhouser: The lesions appeared in May, and from that time they progressed more or less without remission. Essentially they began as vesicles, becoming somewhat purulent and leaving a raw area in the genital area. By September the groin was involved, and during her hospitalization the lesions extended up into the trunk and over the scalp. Some areas around the scalp and the ears were suggestive of seborrhea. The petechial areas that started in the groin had a scale-like coating which could be considered as seborrheic.

Dr. Delp: When this child first became ill in May of last year were vesicular lesions seen only on the genital area, or were there lesions elsewhere on her skin?

Dr. Etzenhouser: None were seen elsewhere.

Gordon Ewy (student): Will you describe the peripheral blood smear on her last admission with special reference to the type and immaturity of the cells?

Dr. Delp: The differential was 60 per cent polymorphonuclear neutrophils (32 per cent filamented, 28 per cent non-filamented), 18 per cent lymphocytes, 2 per cent basophils, 28 per cent monocytes.

Joseph Henning (student): Was a touch preparation of the skin done for histiocytes?

Dr. Etzenhouser: Yes.

Mr. Cashman: Did she have any bone pain at any time during her course?

Dr. Etzenhouser: None was reported.

Bruce Gill (student): What was her fever course?

Dr. Etzenhouser: She was essentially afebrile until late in August. Early in September she developed fever to 103 degrees, and from October 28 she spiked fever almost daily to 104 degrees. There were remissions occasionally, and during one week she had fever of about 101 degrees.

Mr. Gill: What were the blood urea nitrogen values on her first and second admissions?

Dr. Delp: The first BUN was 10.5 mg. per cent; the second was 20.5 mg. per cent.

Mr. Cashman: Was a Coombs' test done?

Dr. Delp: No.

Mr. Gill: What was the reticulocyte count?

Dr. Delp: It was as high as 27 per cent.

Mr. Henning: Did she have facial edema?

Dr. Etzenhouser: There was a fullness about her face, but I do not believe that it could be described as edema.

Mr. Gill: Was this fullness noticeable before corticoid therapy was started?

Dr. Etzenhouser: Yes.

Mr. Henning: Was a serum cholesterol done?

Dr. Etzenhouser: No.

Mr. Cashman: What was the serology on the patient and her mother?

Dr. Etzenhouser: We do not have that report.

Mr. Ewy: Were antibiotics given?

Dr. Etzenhouser: Antibiotics were started after the first week of hospitalization, and continued throughout her course.

Mr. Ewy: Were blood cultures taken on her last admission?

*Although a student at the time of this conference in March, 1961, he, like the others referred to as students, received the M.D. degree in June, 1961.

Dr. Etzenhouser: Blood cultures were taken on numerous occasions during her first admission and at least once on her last admission, but no growth was reported.

Mr. Cashman: Were precipitin or complement fixation tests done?

Dr. Etzenhouser: No.

Mr. Henning: Were stool cultures done at the time she had diarrhea?

Dr. Etzenhouser: Stool cultures were negative.

Mr. Henning: Will you describe the diarrhea?

Dr. Etzenhouser: It was watery and loose with some mucous and occasional flecks of blood, and it was fairly profuse during the week that it lasted.

Mr. Gill: Will you describe the clinical picture before death?

Dr. Etzenhouser: The child was extremely dyspneic and pale. Her pulse was rapid, and her extremities were cool.

Dr. Delp: Was she cyanotic?

Dr. Etzenhouser: She was quite pale, but I do not believe that she was cyanotic.

Mr. Ewy: Was she in a Croupette at that time?

Dr. Etzenhouser: She was in a Croupette with oxygen.

Mr. Henning: Were there any other chest findings?

Dr. Etzenhouser: Rhonchi and rales developed rather suddenly about three hours before death.

Mr. Cashman: Was there any clinical evidence of dehydration?

Dr. Etzenhouser: No.

Mr. Gill: Were skin cultures or gastric washings done?

Dr. Etzenhouser: Cultures of the skin lesions showed non-hemolytic staphylococci, numerous enterococci and E. coli, and the drainage from the ear showed enterococci and E. coli. No gastric washings were done.

Mr. Ewy: Were the lesions described as umbilicated at any time?

Dr. Etzenhouser: Only on the last admission.

Mr. Gill: Were any liver function tests done, any repeat serum proteins or bilirubins?

Dr. Delp: The repeat serum proteins varied only slightly from those on the first admission.

Mr. Cashman: Was there any history of the patient's use of raw milk?

Dr. Etzenhouser: No.

Mr. Henning: Were there any changes in platelets?

Dr. Etzenhouser: The platelet count was consistently low, ranging from 80,000 down to 30,000.

Dr. Delp: May we have the x-rays now, please?

X-rays

Mr. Ewy: A chest film taken on the first admission shows evidence of soft tissue shadows which could confirm the axillary and cervical lymphadenopathy. There is no evidence of bony abnormalities or osteolytic lesions. The costophrenic and cardiophrenic angles are clear, and the lung fields look normal. The heart shadow is not remarkable.

An abdominal film taken on the first admission *(Figure 1)* shows an osteolytic lesion in the wing of the ileum. There is no other evidence of bony abnormalities. There is evidence of hepatomegaly but no splenomegaly.

No bony abnormalities are seen on the film of the skull, and the sella is normal in size.

A film taken on the second admission does not demonstrate any bony abnormalities, and the costophrenic and cardiophrenic angles are clear. A definite infiltrate is seen on the more central area, but peripherally the lungs are clear. There is a suggestion of a mediastinal mass. The heart is of normal size. I interpret these films as showing soft tissue swelling, evidence of osteolytic lesions, and, terminally, pulmonary infiltration.

Dr. Delp: Thank you. Dr. Germann, may we have your comments?

Dr. Donald Germann (radiologist): As has been pointed out, in the margin of the ileum on the right side there is a smooth defect, and there is no bony reaction change or soft tissue swelling around it; it is strictly lytic.

Dr. Delp: Thank you. We are now obligated to make a primary diagnosis of the disease which first brought the child to the physician, the interpretation of the course of the disease and, finally, an explanation for her death. May we have your discussion, please, Mr. Henning?

DIFFERENTIAL DIAGNOSIS

Mr. Henning: This small child had a normal development and was apparently well until the onset of her illness at 11 months of age when she developed lymphadenopathy, fever, pallor and weakness. Two months later she developed hepatosplenomegaly, a petechial rash, anemia and thrombocytopenia. Her hospital course was marked by fever, persistent anemia and progression of the rash, and she developed tonsillitis, otitis and diarrhea. Her illness apparently was stabilized on corticoids, but following her discharge, the rash became generalized. She was readmitted at 15 months of age, and died two days later in respiratory distress.

My differential diagnosis is based on generalized lymphadenopathy, hepatosplenomegaly, anemia, thrombocytopenia and cutaneous lesions, and a rapidly fatal course in a 13-month-old child. Fifty years ago if an infant developed hepatosplenomegaly, anemia, and skin lesions involving her palms and the soles of her feet the diagnosis probably would

have been congenital syphilis. Lymphadenopathy is variable but can be quite marked, and untreated cases may be rapidly fatal. The manifestations of that disease almost always appear before the age of three, and the serology is positive in 95 per cent of the patients over six months of age. This entity can be ruled out because of the age of onset and the lack of typical stigmata. I would prefer to rule it out on the basis of the negative serology, but we have no information in that regard.

Infectious granulomas such as coccidiomycosis, histoplasmosis, and tuberculosis in their disseminated forms may present with many of the signs and symptoms that were seen in our patient. A definitive diagnosis in each of these disease states is dependent upon the isolation of the causative agent by culture or by its demonstration in the tissues. Serological testing could also be helpful. The lack of exposure in endemic areas and the atypism of the subsequent clinical course discounts coccidiomycosis. Histoplasmosis cannot definitely be excluded, but thrombocytopenia and peripheral lymphadenopathy of a marked degree are not usually present in children until late.

Although it is difficult to dismiss tuberculosis the lack of a history of contact, the matted character of the lymph nodes, the acute course, and the patient's response to corticoid therapy militates against that diagnosis.

Patients with lymphoma initially present first with lymphadenopathy. They exhibit fever and anorexia, and ultimately develop a leukemic blood picture with anemia, thrombocytopenia and hemorrhagic tendencies. Lymphoma is uncommon in infants, however, and does not explain our patient's early skin manifestations, thrombocytopenia and anemia. Acute leukemia in children is a rapidly fatal disease characterized by fever, marked prostration, a rapidly developing anemia, and, frequently, hemorrhagic manifestations. Although lymphadenopathy and hepatomegaly are less conspicuous than in the chronic types of the disease, cervical node enlargements may be great in many cases of acute leukemia. The degree of leukocytosis is often misleading. The number and type of the immature forms are more important. As in our patient, children with acute leukemia not infrequently develop necrotic and gangrenous processes of mucous membranes of the mouth and throat. Factors against the diagnosis are the lack of bone pains which are frequently found in the leukemics, the cutaneous lesions, and the lack of significant numbers of immature forms of leukocytes in the early peripheral blood smear. Although the child's illness can be explained by one disease process, it must be remembered that one of the fatal complications of corticoid therapy is an overwhelming viral sepsis with a member of the herpes-varicella group. This secondary complication could help to explain the various skin manifestations seen in our patient. It is not possible to rule it out on the information which is available.

I propose another disease process as a pathological state to explain the infant's death. Histiocytosis-X, a term proposed by Lichtenstein in 1953, describes a spectrum of diseases which include eosinophilic granuloma, Hand-Schuller-Christian disease, and Letterer-Siwe disease. Histiocytosis-X is only one subdivision of a broader classification of the reticuloendothelioses. The majority of these diseases are manifested by disturbances of lipid metabolism and central nervous system derangement, neither of which were seen here. The three idiopathic diseases which make up histiocytosis-X were originally described separately, but they have in common a hyperplastic process of the reticuloendothelial system which results in the formation of granulomas. This spectrum began with Letterer-Siwe disease which is an acute, fatal disease of infants. Hand-Schuller-Christian disease is a chronic disease of young adults, and eosinophilic granuloma is a benign, non-focal destructive bone tumor in older children and adults. The divisions are still called by their original names, and vary primarily in the age of onset and the acuteness of their clinical course.

My primary diagnosis of the patient is Letterer-Siwe disease. Although the first case of this disease was reported by Letterer in 1924 it was not a well described entity until nine years later when Siwe delineated its clinical and pathological characteristics. Letterer-Siwe disease is a rapidly progressive, fatal disease in infants, and it is characterized by generalized lymphadenopathy, hepatosplenomegaly, anemia, hemorrhagic diathesis, osteolytic bone lesions, and cutaneous manifestations. The cutaneous lesions, have variously been described as maculo-papular, seborrheic, yellow, scaly, vesicular, or any combinations of these. The infants are extremely susceptible to secondary infection, and otitis, and pharyngitis and pneumonitis are common. Pneumonic infiltration of the lung parenchyma by histiocytes as well as incroachment of the respiratory passages by enlarged mediastinal lymph nodes are often seen. This compromise of the respiratory function together with the anemia may explain the patient's tachycardia and tachypnea. A superimposed pneumonitis and possible septicemia resulted in cardio-respiratory collapse and death. Our patient exhibited all of the typical manifestations of Letterer-Siwe disease.

It is interesting to note the patient's terminal blood picture, especially the monocytosis. In a review of the literature there were five cases of Letterer-Siwe disease terminating in acute monocytic anemia. In postulating Letterer-Siwe disease as a primary diagnosis it is well to remember that this disease and acute leukemia may exactly mimic each other. The

final definitive diagnosis is based on histologic examination, and even then a conflicting diagnosis may occur.

CLINICAL DISCUSSION

Dr. Delp: Thank you, Mr. Henning. What is your diagnosis, Mr. Cashman?

Mr. Cashman: I believe that the skin manifestations could best be explained by Letterer-Siwe disease.

Dr. Delp: Mr. Gill?

Mr. Gill: My diagnosis is Letterer-Siwe disease, possibly terminating with septicemia or viremia.

Dr. Delp: Mr. Ewy?

Mr. Ewy: Letterer-Siwe disease.

Dr. Delp: Mr. Cashman, assuming that your diagnosis is correct, how do you explain the patient's lymphadenopathy? Was this a neoplastic process?

Mr. Cashman: It is believed by some that Letterer-Siwe disease is a neoplastic process involving the histiocytes and reticulocytes. Multiplication of these cells in the lymph nodes could adequately explain the lymphadenopathy. The typical pathological picture is enlarged granulomas with the histiocytes in the lymph nodes throughout the body.

Dr. Delp: What is your explanation for the low platelet count, Mr. Gill?

Mr. Gill: A low platelet count is typical of Letterer-Siwe disease. Hypersplenism or an enlarged spleen may also account for the decrease in platelets. One could also postulate the bone marrow maturation arrest or abnormality in the platelets, but I do not believe that that was the reason for it.

Dr. Delp: What is your explanation for the reticulocytosis, Mr. Ewy?

Mr. Ewy: I believe that it was just a compensation process for the marked anemia which was probably on the basis of a hemolytic process. The histiocytes also phagocytize the red cells.

Dr. Delp: What produced the hemolysis?

Mr. Ewy: I believe an immunological process cut short the red cell life.

Dr. Delp: What is your explanation for the bone lesions, Mr. Henning?

Mr. Henning: Bone lesions usually show a granulomatous process. Although I can not explain the exact mechanism, this is a typical lesion which is also found in that disease.

Dr. Delp: The child was placed on corticoids some time during August or September. What are your comments about the part that corticosteroids might have played in her subsequent course, Mr. Cashman?

Mr. Cashman: As far as is known at the present time, the treatment for Letterer-Siwe disease consists of corticosteroids, and favorable responses have been reported with that type of therapy. It is stated in the protocol that her fever did go down and her hemoglobin went up. I believe that clinically she showed some improvement on corticosteroid therapy. As has been mentioned in connection with leukemia, several complications of corticosteroid therapy could have been present here, but I prefer to explain it on one logical basis of a pathological process.

Dr. Delp: The skin lesions seen here played an important part in the patient's disease. What are your comments about these skin lesions, Mr. Gill?

Mr. Gill: Many types of lesions are described with our primary disease process, and these may vary and involve the palms and soles of the feet. They may be petechial and purpuric. One should not lose sight of the fact, however, that cases often develop a viral disease, while on corticosteroids. A rash may have developed from viremia, for example, varicella or vaccina, which would account for vesicular lesions.

Dr. Delp: Mr. Ewy?

Mr. Ewy: I agree with that, especially since these lesions were described at one time as being umbilicated.

Dr. Delp: Mr. Henning?

Mr. Henning: There is the possibility that these lesions could be unrelated to the primary disease process.

Dr. Delp: What is your explanation for the diarrhea, Mr. Cashman?

Mr. Cashman: One explanation is the basic pathologic process which has been postulated. Infiltration with histiocytes occur in lymphoid tissue all over the body, including the intestinal tract. Ulcerations of the intestinal tract could cause irritation and diarrhea, although there was not much evidence of bleeding into the gastrointestinal tract. In addition, she received antibiotics for the pharyngitis and otitis, and that could have caused a change in the gastrointestinal flora and diarrhea.

Dr. Delp: Mr. Gill?

Mr. Gill: There could have been an overgrowth of staphylococci.

Dr. Delp: Dr. Wenner, may we have your comments?

Dr. Herbert A. Wenner (research professor of pediatrics): When I first saw the patient there were lymphadenitis in the groin and large lymph nodes in the neck. The lymph nodes were biopsied with the pathological report of granulomatous reaction but not specific in any particular details. At that time I was uncertain of the diagnosis because monilia had been cultured on the lesions in the vagina, in the area of the breakdown of the lymph nodes, and at the site of the biopsy. The patient had a normal white count, and repeat blood cultures were negative. She had a spiking temperature which would return to normal with various antibiotics, but while still under treatment would rise again.

The second time that I saw the baby my impression

was that this was a generalized reticuloendotheliosis because the rash on the skin had become much more apparent, and some of the potential characteristics of Letterer-Siwe disease, or generalized reticuloendotheliosis, had developed. Another lymph node was biopsied on the basis of that impression, and the results confirmed the clinical impression that this was a reticuloendotheliosis, and, at least, a granulomatous reaction existed with it. The bone lesions were not initially seen.

The baby did fairly well on corticoids while under antibiotic therapy, and on November 30 it was decided to let her go home to the grandparents for Thanksgiving. On that day another patient in the early stages of chickenpox was admitted. We were cognizant of the fact that that imposed a risk on the baby, a point which further forced my hand to let her go home. The mother later told me that the two children were at the opposite ends of the floor and that the only contact was in passing by the door of the baby who had the chickenpox. We asked the mother to be on the alert, however, and in about 12 days she brought the baby back to the hospital with lesions that supposedly were early lesions of chickenpox. These lesions became quite evident within the next two days, and it was apparent that this was a life-threatening affair for her. She was given gamma globulin on the second day after the earliest vesicles appeared, but she died of generalized chickenpox.

Dr. Delp: Could we have your comments, please, Dr. Frenkel?

Dr. Jack Frenkel (pathologist): When I saw the baby and the biopsy I thought that because there were numerous necrotic areas in the lymph nodes that there could be a reasonable chance that this was not a neoplastic disease, but an infectious disease. Therefore, it was worthwhile to attempt to isolate something from it which could perhaps be treated specifically. Consequently, animals were inoculated and treated with cortisone to enhance any possible agent which might be there or depress the immunogenesis, but nothing was isolated. In reviewing the literature I found that Letterer-Siwe disease occasionally gives rise to moderately extensive areas of necrosis, and I acquiesced in that diagnosis.

Dr. Delp: Thank you. Dr. Weber, you saw the patient the day before she died. May we have your comments, please?

Dr. Robert Weber (internist): At the time I saw the baby the diagnosis of Letterer-Siwe disease was fairly well established, and the problem was primarily that of the disseminated varicella, or chickenpox. This child had two reasons for increased susceptibility, the primary disease probably being the more important, but the presence of the corticosteroids undoubtedly increased the danger from the chickenpox. That has even been reported in normal children. In my opinion, this is one of the most important lessons to be learned here. Any child who is on corticosteroids is in danger if exposed to chickenpox because then it becomes a disseminated disease. The viremia becomes extremely dangerous, and the mortality rate is enhanced tremendously. I would be surprised to find that this child had a chickenpox pneumonia as a complication, although the protocol certainly makes it seem likely. That complication has been almost exclusive in adults, but with the corticosteroids, I wonder whether it could have been a possibility at the post mortem.

Mr. Cashman: What was the dose of corticoids?

Dr. Delp: The dose was decreased considerably; I believe it was cut in half.

Mr. Cashman: Did she show any improvement after the decrease in dosage?

Dr. Delp: No, because she had been exposed to chickenpox. Now if there are no more questions, we will have the pathological report.

PATHOLOGICAL REPORT

Dr. Frank Mantz (pathologist): It seems that there is little left to do in this case but to confirm everybody's diagnoses. It is my belief that this can best be done by a review of the biopsy of the lymph node which was obtained in life. There we observed a diffuse proliferation of rather wild and distinctly abnormal reticuloendothelial cells replacing the normal architecture and showing evidence of rather striking mitotic activity. Associated with this was a somewhat extensive degree of giant cell formation, numerous chronic inflammatory cells and many eosinophiles.

There were also many broad areas of necrosis, similar to those described by Otani,[1] resulting in a somewhat granulomatous appearance. I believe that this is certainly the classical histological appearance of Letterer-Siwe disease.

I do not intend to indulge in the usual polemics which follow a discussion of this particular process, but I would like to establish my personal feeling that this is a quasi neoplastic proliferation of reticuloendothelial cells closely related to eosinophilic granuloma and Hand-Schuller-Christian disease, and that mutations between these various forms do occur.

At autopsy there was rather striking evidence that the lesion had been considerably affected by therapy. There had been complete reduction of the peripheral lymphadenopathy and traces of the disease could be found only in the mesenteric lymph nodes, the lymphoid tissue of the gastrointestinal tract, the retroperitoneal lymph nodes, and possibly in the lung, the spleen and the bone marrow.

The superior mesenteric lymph nodes offered the only area where we could satisfactorily confirm the diagnosis. At other sites the disease was altered beyond recognition, a feature which I hope to bring out

Figure 1. X-ray taken on the first admission.

from another angle. In these lymph nodes there were residual large, abnormal reticuloendothelial cells. They were markedly reduced in number permitting recognition of the underlying normal lymph node architecture.

The individual cells were quite different from those we observed previously. There was no evidence of mitotic activity. The tumor elements were quite swollen and frequently appeared vacuolated. They did not, however, contain fat. I must assume that this is an effect of adrenocorticosteroid therapy, producing regression by inhibition of mitosis and subsidence of the lesion by attrition of abnormal cells.

Within the lymph nodes, as well as the liver and the spleen, there was abundant evidence of hemosiderosis. Within the liver and spleen this was associated with extra medullary hematopoiesis, and I must assume that there was indeed a hemolytic process. This feature is quite common in abnormal proliferative disorders of the reticuloendothelial system of all types.

The most interesting feature of the autopsy related to the terminal illness. Observed upon the skin were a multitude of vesicles *(Figure 2)* apparently occurring intra epidermally and apparently of varying ages. Some were quite isolated, fresh, and consisted of tiny dewdrops surrounded by an erythematous base. Others were clustered and had become confluent with evidences of hemorrhage, early ulceration and crusting. Within them there was ballooning degeneration of the epithelial cells associated with marked nuclear abnormality and cellular multinucleation. Within these cells there were large and rather characteristic intranuclear bodies associated with contracture of the nucleoplasm to produce the appearance of what is known generally as Cowdry type A inclusions *(Figure 3)*. These inclusions are rather typical of varicella but certainly do not permit us to make a specific diagnosis since the viral inclusions of varicella, herpes zoster and herpes simplex are practically identical morphologically. The gross appearance and distribution of the lesions, however, and the history of exposure certainly make the lesions those of varicella beyond peradventure of doubt.

Striking changes were seen in the lungs. These were massively enlarged but showed no gross evidence of consolidation. There were punctate macular areas of discoloration immediately beneath the pleura which represented irregular foci of somewhat hemorrhagic, fibrinous and necrotizing pneumonitis. Similar lesions were also scattered throughout the central areas. Small intranuclear inclusions were present here as well, involving the septal cells *(Figure 4)* and large desquamated alveolar cells. I believe it to be highly significant that there was no evidence of inflammatory infiltration.

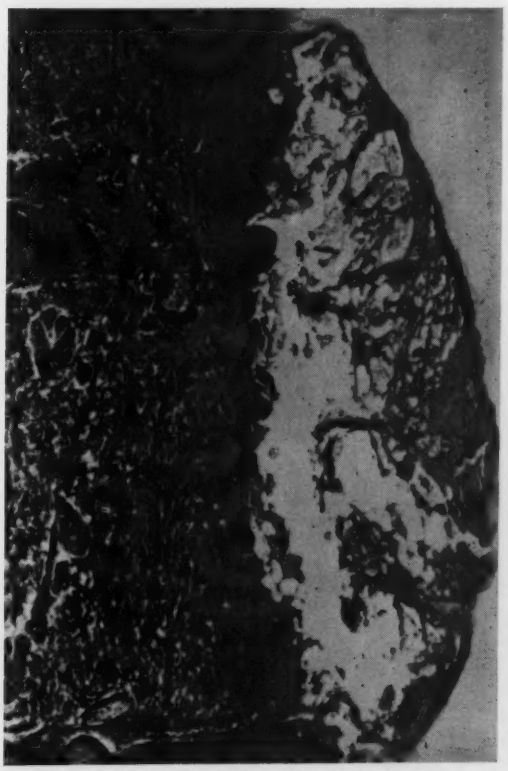


Figure 2. Intraepidermal vesicle of varicella.

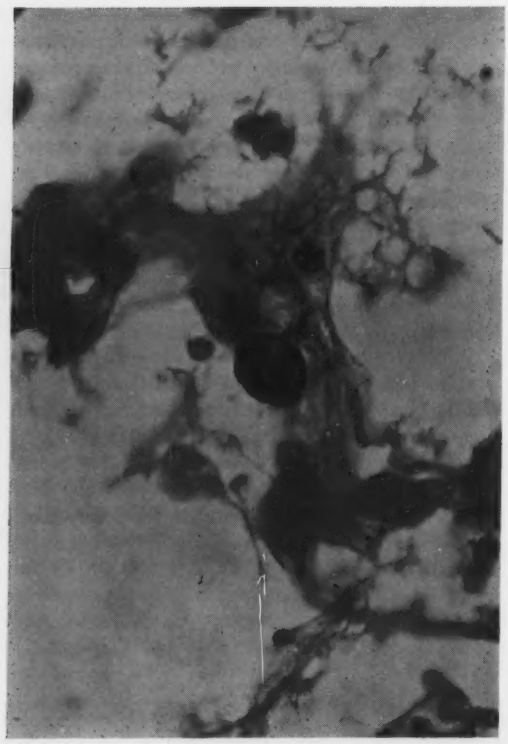


Figure 3. Intranuclear varicelliform inclusions in acantholytic cell of epidermis.

Lesions similar to those noted in the skin were present in the hypopharynx and upper esophagus. Some of these were early and showed the profound cellular alterations produced by virus infection including cellular gigantism, cellular irregularity, individual cell necrosis and amitotic division with formation of syncytia. Scattered inclusions could be observed in occasional cells throughout the remainder of the gastrointestinal tract, but no additional specific lesions were present.

It is of some interest that virus infection extended throughout the biliary tree. Intranuclear inclusions were observed frequently within the bile ducts many of which appeared necrotic. Occasionally this resulted in total loss of the lining epithelium with bile diffusion and the production of bile granulomata. Associated with this were focal areas of necrosis extending from portal areas to involve the peripheral portion of lobules. Adjacent to the necrotic areas many of the polygonal cells were found to contain viral inclusions *(Figure 5)*. The absence of inflammation once more was striking.

To return to the reticuloendothelial system, we made the statement that, other than in the upper mesenteric lymph nodes, the diagnosis of Letterer-Siwe's disease could no longer be achieved. In this regard the lymphoid follicles and Peyer's patches within the intestinal tract are of interest showing evidence of remote necrosis and ulceration which since had healed with epithelialization of the surface. I believe that this fits well with the history of diarrhea. The underlying lymphoid tissue, as well as the lymph nodes in the retroperitoneal and peri-iliac areas, and the spleen showed profound necrosis. Here the degree of destruction was far in excess of that which was observed originally in the superior mesenteric nodes *(Figure 6)*. This can be explained in part by a tremendous population of Letterer-Siwe type cells showing abundant intranuclear viral inclusions *(Figure 7)*. There was also evidence of rather profound proliferation of capillaries throughout the area, each capillary apparently plugged by either proliferating or swollen endothelial cells infected with virus. This invites consideration of a predilection of varicella virus for young and proliferating tissue. Furthermore, a specific oncolytic effect is suggested since necrosis of tumor was evident only where virus infection was found.

The spleen showed threefold enlargement and contained foci of minute necrosis confined to the follicles

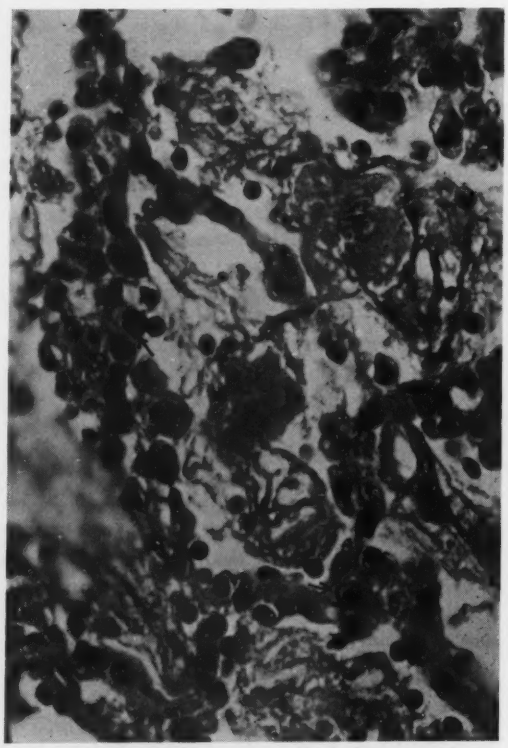


Figure 4. Varicella pneumonia. Varicelliform inclusions in alveolar septal cells.

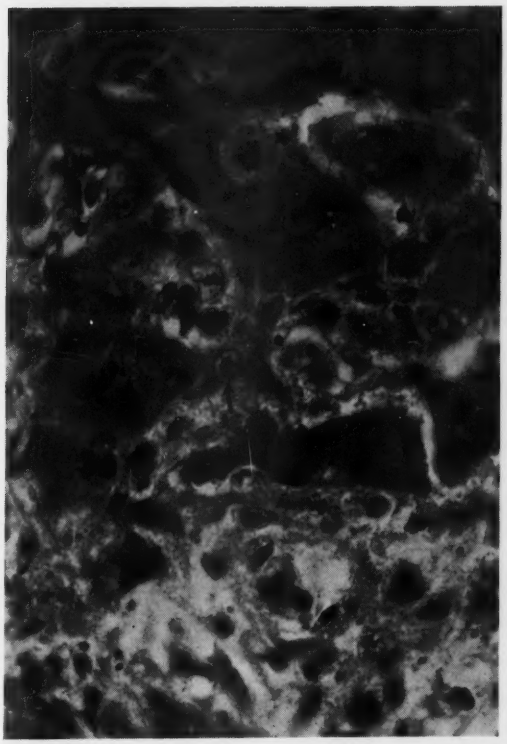


Figure 5. Varicella hepatitis. Periportal necrosis with varicelliform inclusions in nuclei of polygonal cells.

and along the trabeculae. Viral inclusions were present in the necrotic masses.

The bone marrow was examined at several sites and was found to be somewhat hypocellular with multiple focal areas of necrosis. Under high power we observed within large reticuloendothelial cells the presence of classical varicelliform inclusions. In the magakaryocyte there was marked nuclear alteration from which I suspect invasion by the virus.

As far as the remaining organs were concerned, a careful study which included the brain, adrenals, pancreas and the kidneys showed no evidence of viral infection except within a parathyroid where one minute area of necrosis was evident and scattered viral inclusions could be seen.

In summary, I believe that the basic morbid process was Letterer-Siwe disease which had regressed under the stimulation of cortisone. In the course of this favorable response, the patient unfortunately developed chickenpox. Presumably because of reduced resistance inherent to cortisone therapy,[2, 3] widespread dissemination occurred. This distribution and morphology is considered typical.[4] I would like to point out that fatal varicella is a rare phenomenon, not exceeding 0.4 per cent in any series. We are quite aware that fatalities of this nature occur in usually benign viral diseases, but since there have been two similar cases, one of varicella and one of disseminated herpes simplex in patients previously the subject of these conferences.

Of extreme interest to me is the alteration observed where virus apparently invaded the Letterer-Siwe tissue suggesting a marked oncolytic effect. This reminds me that in the recent past viral therapy was a popular area for research among oncologists.[5] West Nile virus and 101 virus were found particularly capable of producing necrosis in animal tumors as well as in tumors of terminal human carcinoma patients.

Dr. Delp: Are there any questions of Dr. Mantz?

Mr. Gill: Was there any marked degree of phagocytosis of the erythrocytes?

Dr. Mantz: Careful search for erythrophogocytosis failed to show this process.

Mr. Cashman: I wonder whether the pathologist found any explanation for the marked increase in the white cell count toward the last, and whether the terminal event would be involvement of the bone marrow.

Dr. Mantz: I infer from the bone marrow changes

(Continued on page 323)

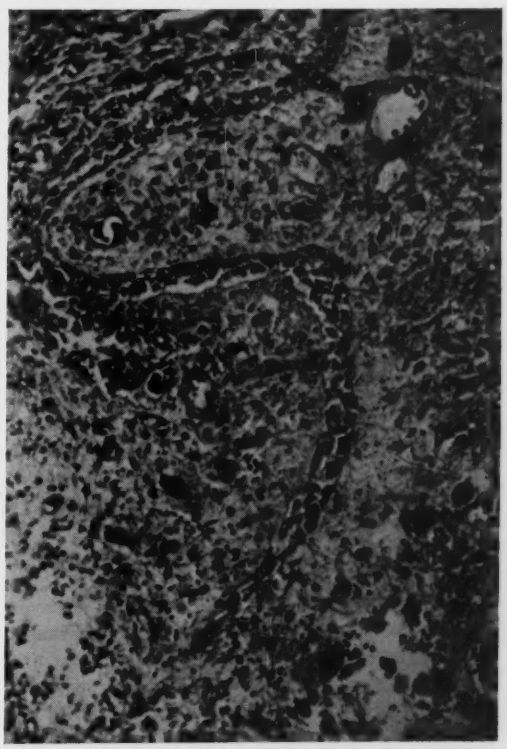


Figure 6. Necrosis of lymph node altered by Letterer-Siwe's disease and showing marked endothelial proliferation.

Radiology

Case of the Month

Edited by COLVIN AGNEW, M.D., *Kansas City*

Radiologic Description

Prominent, coarse, wavy trabeculae have replaced the normal delicate fine uniform pattern of the pubic and ischial rami and have extensively altered the texture of both iliac bones. There has been enlargement of the ischial and pubic rami. While overgrowth of bone has not been demonstrated of either iliac bone, there has been an overall increase in density. The lumbar vertebral bodies and femora are essentially normal. There does not appear to be any involvement of the sacrum.

Osteitis deformans (Paget's Disease) can hardly be mistaken for anything else when it presents this radiologic appearance. Paget's Disease in its less classic appearance may, at times, be difficult to distinguish from metastatic carcinoma of the prostate.

Clinical Summary

A sixty-five-year-old man was admitted with abdominal distress and with a tentative diagnosis of duodenal ulcer disease. The film of the abdomen (*Fig. 1*) was made before the barium was administered. He had no symptoms relative to the changes seen in the bone. His abdominal complaints have been proven to be due to pancreatic carcinoma.

Discussion

Osteitis deformans is rarely seen under the age of thirty and the greatest incidence is between forty and fifty. The sacrum, lumbar spine and femurs are most frequently involved with the skull, sternum and pelvis next most affected. It may involve one bone or several.

Radiologic findings are quite characteristic. When the shafts of long bones are involved, these are always thickened and expanded. The cortex loses its homogeneous density and the trabeculae appear in the region of the usual cortical density. The marrow cavity is partially or completely obliterated by a wavy thick bony meshwork with radiolucent interstices. These longitudinal bony striae appear grouped into bunches or wavy strands, especially in the neck of the femur and the pelvis. Long bone shafts are bowed later due to softening. Although the density is increased, the bone is actually softer than normal. Small cyst-like areas may be seen between these heavy wavy trabeculae with extensive new bone formation, particularly in the pelvis. This may resemble carcinoma of the prostate in certain instances and in certain stages of the disease, particularly when the pelvis is involved.

Characteristic changes with metastatic carcinoma of the prostate resemble Paget's Disease very little. The usual mixed osteolytic and osteoblastic lesions from metastatic carcinoma of the prostate produce circular areas of increased density of various sizes. Early, they may involve only small parts of the bone but as they later become confluent and are usually multiple, these extensive changes may resemble some forms of Paget's Disease. The bone destruction is seldom visible and is masked by osteoblastic reaction. There is no tendency to form longitudinal striae as seen in Paget's Disease. There is no tendency for the bone to enlarge as is regularly seen in Paget's Disease. In border line cases it may be difficult to distinguish between metastatic carcinoma of the prostate and Paget's Disease.

Frequently blood chemistry determinations will easily swing the pendulum to the proper diagnosis. Carcinoma of the prostate with bony metastasis usually produces an elevated serum acid phosphatase while Paget's Disease does not. Both may produce elevated serum alkaline phosphatases. Most references state that Paget's Disease is about equally divided between the sexes. There have been reported instances of multiple cases within a family, both in the same and different generations.

Complications of Paget's Disease include fracture, pseudo fracture, and malignant changes. Fractures usually are transverse and heal rapidly and do not re-fracture easily. Pseudo fractures appear as a transverse zone of decalcification running from the periphery to the convex border to the medulla. They may be single or multiple.

Sarcomatous degeneration represents the most serious complication of Paget's Disease. It has been estimated that nearly 40 per cent of all osteogenic sarcoma may occur in association with Paget's Disease.[2]

The original description leaves little to be desired. Sir James Paget considered the following to be the chief characters of the "infection." "It begins in middle age or later. It is very slow in progress and may continue for many years without influence on the general health and give no other trouble than

Figure 1. Extensive alteration of normal trabecular pattern and bone density of the pelvic bones in an asymptomatic man.

those which are due to the changes of shape, size and direction of diseased bone. Even when the skull is largely thickened and all of its bones exceedingly altered in structure, the mind remains unaffected. The disease affects most frequently the long bones of the lower extremities in the skull and is usually symmetrical. The bones enlarge and soften, and those bearing weight yield and become unnaturally curved and misshapen, suggesting the proposed name, 'Osteitis deformans.' The spine, whether by yielding to the weight of overgrown skull or by changes in its own structure may sink and seem to shorten, with greatly increased dorsal and lumbar curves; the pelvis may become wide, the necks of the femora may become nearly horizontal, but the limbs, however misshapen, remain strong and fit to support the trunk. In its earlier periods and sometimes through all its course the disease is attended with pains in

Several recent issues of The JOURNAL have included a case report in the field of radiology. The Editorial Board feels that these case reports are interesting and instructive, and invites contributions from radiologists anywhere in the state. Though it may not appear each month, it will appear whenever suitable material is available.—*Ed.*

Medical Societies.

ROYAL MEDICAL AND CHIRURGICAL SOCIETY.

THERE was an unusually large attendance at the meeting of this Society on the 14th inst., when a paper upon a form of chronic inflammation of bones (osteitis deformans), by Sir James Paget, President, was read and discussed, the speakers being Sir William Gull, Mr. Brudenell Carter, Mr. Barwell, and Dr. Goodhart.

Figure 2. Paragraph 1, from the account of Sir James Paget's original description.

the affected bone—pains of widely various severity, and variously described as rheumatic, gouty, neuralgic, not especially nocturnal or periodical. It is not attended with fever. No characteristic conditions of urine or feces have been found in it. It is not associated with any constitutional disease unless it be cancer, of which three out of the five cases recorded in the paper were the subject."

Acknowledgement

The clinical material was presented by Melvin Masterson, M.D., Department of Radiology, KUMC.

References

1. Archer, V. W., The Osseous System, Year Book Publishers, 1945.
2. Brailford, J. F., Radiology of Bones and Joints, Williams & Williams Company, Baltimore, 1948.
3. Paget, J., On a Form of Chronic Inflammation of Bones (Osteitis deformans); The Lancet, Vol. 2, Page 714-716, 1876.

Torticollis

(Continued from page 306)

that he is interviewing and being interviewed for a variety of sales jobs.

Editor's Note: This article was submitted to THE JOURNAL for publication in March, which is the University of Kansas Medical Center issue. Due to space limitations at that time, THE JOURNAL was able to publish only half of the articles submitted.

The remaining papers will appear in the upcoming issues.

THE JOURNAL would like to thank these authors for being so patient while waiting for their article's publication.

Tell a man something is bad, and he's not all sure he wants to give it up. Describe it as stupid, and he knows it's the better part of caution to listen.—*David Seabury*

We would all rather be in the company of somebody we like than in the company of the most superior being of our acquaintance.—*Frank Swinnerton*

Hereditary Nephritis

HUGH D. GREER, III, M.D., ***New Hampshire***

RECENT LITERATURE indicates that hereditary nephritis is an established, but ill-defined entity. Thirteen reports of hereditary nephritis involving three or more generations were found in a search of the literature from 1881 when the first kindred was studied. It has been reported under the following designations: "Idiopathic" or congenital and hereditary family hematuria; hereditary familial congenital haemorrhagic nephritis; hereditary interstitial pyelonephritis; congenital hereditary hematuria; hereditary hematuria, nephropathy and deafness; and several other variations. It is the purpose of this paper to summarize the work which has been done and to attempt a clinical, pathological, and genetic correlation, in order to establish reasonably accurate criteria for determining what constitutes hereditary nephritis.

The Problem

The study of a relatively rare, hereditary disease in man is complicated by his slow rate of reproduction and migratory habits. One of the earliest kindreds reported was investigated by three men at different times over a period of 25 years, and the investigation spanned two continents. It is fortunate that this and other clans in the total study have been more than commonly prolific.

Case reports span a period of nearly eighty years, from 1881 to the present. During this period, advances in diagnostic technique have rendered much of the earlier work incomplete, though not invalid. In the older investigations, tissue examination has been inadequate or totally lacking, and other techniques, such as audiometry, which would be particularly valuable in this disease, are not widely used even at present. Fortunately, much genetic information can be obtained in retrospect, and the errors of memory and hearsay tend to be minimized by statistical evaluation. Indeed, one of the best genealogical investigations in the literature was among the first reported, and has been reinforced by the efforts of two later investigators who were able to follow the family for a quarter-century.

The Clinical Disease

The clinical picture in this disease is by no means uniform, but two observations appear with sufficient regularity to require that it be investigated as a single entity: renal disease culminating in early death, particularly in males; deafness. This association is reported by Guthrie, Perkoff et al., Sohar, Reyersbach and Butler, Sturtz and Burke, Goldbloom et al., Robin et al., Goldman and Haberfelde. The types of renal disease reported fall into two fairly distinct clinical groups:

1. Those in which the disease resembles chronic glomerulonephritis, with hematuria as the outstanding clinical manifestation. This group is fur-

This is one of a group of theses written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be best by the faculty at the school. Dr. Greer is now at the Mary Hitchcock Memorial Hospital, Hanover, New Hampshire.

ther characterized by onset in infancy or early childhood, and by early death in males.

2. Those in which the prominent finding is pyuria, and in which the clinical course is that of chronic pyelonephritis.

These groups are not mutually exclusive. They are divided here only because they are so classified in the literature.

The papers of Guthrie, Sturtz and Burke, Reyersbach and Butler, Goldbloom and Haberfelde, are notable studies of the first group. Guthrie's extensive study was first reported in 1902 with the title " 'Idiopathic' or congenital hereditary and family hematuria." In the kindred investigated, hematuria and albuminuria were prominent clinical features of the syndrome, but pyuria was not. Intermittent attacks of hematuria could be induced in the majority of affected individuals by eating black currants, drinking claret, and the ingestion of a number of other more common viands. In the family studied by Reyersbach, hematuria was often precipitated by any fever. Recent authors have emphasized the presence of tubule casts, and Eason, conscious of the resemblance of this disease to glomerulonephritis, suggested in 1924 that hereditary nephritis was a more appropriate term for the disease. Sturtz and Burke, in 1956, set down rigid criteria for this diagnosis, and insisted that the presence of pyuria, or of renal deaths in females, should exclude a particular case or kindred from this designation. In the reports of this glomerulonephritis-like group, the lethal course of the disease in males is striking. In Reyersbach's series, there were no female deaths, and no affected male lived beyond 29 years.

Perkoff et al. in 1951 were the first to report a large kindred in which the primary clinical findings were chills, fever and pyuria, consistent with pyelonephritis, but in which the other criteria of heritability and deafness were prominent. Their pathological studies were the first to show that although either hematuria or pyuria might be the more clinically prominent, the finding of "foam cells" in severely scarred kidneys at post-mortem examination in both groups suggested that the two clinical types were different aspects of the same entity. It is interesting that study of older reports in which the finding of hematuria was emphasized shows that pyuria occurred often, both in the same subject and in other members of the same kindred. Recent reports by Mamou and Goldman and Haberfelde also fall into the pyelonephritic category.

Deafness, first reported by Guthrie, is seen with such regularity in this syndrome that its absence must exclude a kindred from consideration. It is nerve deafness, apparently cochlear, and recent studies indicate that it is present to some degree in all affected males and many affected females. The most severe hearing loss falls in the 500-4000 cycles per second range and may vary from 20 decibels to total deafness. Where audiometric studies have been done, it appears that while nephritis can occur without measurable deafness, the converse is not true. The nephritis usually appears first however, and may have progressed to a chronic asymptomatic stage by the time the deafness becomes manifest. The severity of the nephritis and the deafness seem not to be correlated with each other. This is particularly true of females, who can progress to total deafness without suffering severe clinical renal disease. The evaluation of this aspect of the disease depends on thorough and consistent audiometric testing, and this is lacking in most published studies. In Perkoff's excellent series, the picture is clouded by the introduction of otosclerosis into the clan by marriage in the II generation. Pathological studies, where available, have contributed little to this aspect of the disease.

Pathology

Early investigations included little or no tissue examination. The first necropsy was reported by Alport in 1927. Two brothers who had suffered from hematuria died in early adulthood within a year of one another. Post-mortem examination of one of the men showed what is described only as "a small white kidney." Because only these two members of the family were reported, and because there was no associated deafness, it is doubtful that this family can properly be considered as having had hereditary nephritis.

In 1954, Reyersbach and Butler reported a kindred of four generations and 77 souls, of which 23 had hematuria. A kidney biopsy was done on a two-year-old male of this clan who had manifested gross hematuria since birth. The biopsy showed no lesions save red blood cells in the tubules. The child continued to have hematuria, exacerbated by any fever, but of decreasing severity, and developed bilateral nerve deafness at six years. Further investigation of the same kindred revealed that a brother had died of chronic renal failure in 1940, at age thirteen. He had been deaf since age nine. Autopsy of this brother revealed shrunken kidneys with a combined weight of 100 grams. Their surfaces were peppered with yellow-orange spots which on section were related to yellow streaks which replaced most of the cortex. Microscopically, these streaks were clusters of lipid-filled cells with the general configuration and size of convoluted tubules. No tubule remnants could be found in these clusters and the authors speculated that the foam cells were fat-laden macrophages. Histochemical study showed the cells to have a high content of cholesterol and phospholipid. There was interstitial

fibrosis and lymphocyte infiltration without evidence of abscess formation. The glomeruli were strikingly involved with the characteristic lesions of chronic glomerulonephritis, including scarring and crescentic proliferation of Bowman's epithelium. Pathologic diagnoses were: (Hereditary renal disease); chronic glomerulonephritis; interstitial nephritis.

Perkoff et al. in 1951 reported an autopsy on an adolescent male in their study, deaf, who had died in uremia. The kidneys were contracted. The glomeruli were considered normal. There was a moderate diffuse infiltrate of "round cells" throughout the cortex. Dilated collecting tubules containing a "cellular" precipitate were noted, and there were many small focal abscesses in the cortico-medullary junction. These authors published a follow-up study on the same kindred in 1958, in which they presented the anatomic findings in four renal biopsies and two necropsies. Biopsies on two adolescent girls showed only slight thickening of the basement membranes in Bowman's capsule, one or two hyalized glomeruli, and a few blood cells in the tubular lumens. No inflammation was noted. Biopsies on two women of 28 showed similar changes, plus focal tubular atrophy and interstitial fibrosis, and in one subject, focal tubular and interstitial lipid deposits. Post-mortem examinations of the kidneys of two older subjects, a 44-year-old male and a 62-year-old female, showed "diffuse cortical scarring and large foci of interstitial foam cells." Glomerular changes were more pronounced than in the younger patients, and included hypercellularity, crescent formation, and hyalinization. Very little evidence for infection was found, although E. coli was cultured from the kidneys in one case. The authors suggested that these findings might represent the different stages in the progression of a single disease with aging. It is noteworthy that the most prominent clinical finding in these patients, and in the kindred, was pyuria, hematuria being next in frequency.

The middle and inner ears of one of these patients were examined at autopsy, but the examination was considered to be technically inadequate, and only minimal hyperostosis of the tip of the cochlea was noted. The organ of Corti was not examined.

Goldbloom et al. in 1957 reported nephritis in three siblings of a Hebrew family. The father was unaffected, but the mother, although asymptomatic, showed hypertension, albuminuria, pyuria, and hematuria. Two male children died at ages five and ten. The youngest child was found at autopsy to have evidence of severe chronic pyelonephritis with large foci of interstitial lipid-laden "foam cells" similar to those described above. A few sclerosed glomeruli were noted. Testis were absent. The second child manifested albuminuria, pyuria, hematuria and blood pressure of 145/108 at age seven. Nerve deafness was noted at that time. He died in uremia at age ten. Post-mortem examination of his kidneys showed chronic pyelonephritis without evidence of glomerular involvement. A few scattered foci of foam cells were found. The oldest child was eighteen years at the time of this report. He had had hematuria, albuminuria, cylindruria and pyuria since eighteen months of age, and had had bilateral nerve deafness since age eleven. He had bilateral anterior subcapsular cataracts. Blood pressure was 140/100 and urea clearance was 30-40 per cent of normal.

Finally, in 1959 Goldman and Haberfelde reported a kindred of five generations in which six males had died of nephritis, five members have clinical glomerulonephritis, and five have intermittent albuminuria. Three biopsies and two necropsies are available. Biopsies were done on two females, aged seven and ten, with similar histories of glomerulonephritis. Minimal but definite abnormalities, consisting of increased cellularity of glomeruli, a few hyalinized glomeruli, and red cells and casts in the tubules, were nearly identical in both cases. One of these children had a bifid kidney on one side and a double ureter on the other. A 48 year-old male, who had suffered from diabetes for seven years, had a renal biopsy which showed chronic pyelonephritis and diabetic glomerulosclerosis. Two males, aged 43 and 36, died in renal failure with similar pathologic findings. The kidneys were small and the capsules adherent. The subcapsular surface was granular and mottled yellowish-tan, with scattered dark red cysts of 0.5 to 1.0 millimeters in diameter. The cortico-medullary junction was distinct, but the cortex was much reduced in thickness and infiltrated with tiny yellow spots. Microscopically, there was severe diffuse nephritis involving all elements. Nearly all glomeruli showed varying degrees of damage, consisting of scarring, pericapsular fibrosis, crescent formation, and cellular proliferation of the capillary tufts. There were dense focal areas of scarring and lymphocyte infiltration suggestive of chronic pyelonephritis. There was marked arterio- and arteriolo-sclerosis. Scattered foci of interstitial fat-laden macrophages were found in one case.

Inheritance

The evidence for genetic transmission of this disease is overwhelming. Its high incidence in each kindred reported suggests that the gene has high penetrance. Other aspects of the process of transmission are not so clear. There are two apparent mechanisms of transmission.

In the families reported by Dickinson, Mamou, Robin et al., and Goldman and Haberfelde, transmission appears to be consistent with that of a simple Mendelian dominant. An affected parent transmits the gene equally well to either sons or daughters, who

in turn are able to transmit the disease to offspring of either sex. It must be remembered that in all series males are more severely affected, and that female deaths from the disease are relatively rare. In Mamou's report, however, four of five affected females succumbed to the disease, and there were no male deaths. The possibility that this gene exists as a recessive, which can cause sub-lethal disease in the heterozygous combination, and is lethal when homozygous, analogous to Hemoglobin SS disease, can be ruled out by the observation that asymptomatic carriers of the disease have consistently been able to transmit lethal disease to their offspring through random mating. Analysis of Perkoff's kindred for dominance is as follows:

Total Subjects	*Affected Subjects*	*Carriers*	*Total*	*Expected*	*Incidence*
202	74	16	90	101	44.5%

Unrecognized carriers in generation IV are not included. Their eventual recognition and inclusion might bring the incidence up to the expected fifty per cent.

There are a number of kindreds in which sex-linkage must play a part. These are reported by Kidd, Guthrie, Perkoff et al., Sohar, and Reyersbach and Butler. In these papers the affected males who had inherited the trait from their mothers transmitted it almost exclusively to their daughters, who were in turn able to transmit the disease only to their sons. This implies that the trait is inherited by the affected male on an X chromosome. He cannot therefore transmit it to male offspring. Had he received the defective gene on the Y chromosome from his father, the reverse would have been true. In the theory of sex-linkage, exceptions to male-female-male transmission can only be explained by the crossing-over of a dominant gene located on the homologous part of the X-Y chromosome. Exceptions do occur in three of the papers above, and they are extensively analyzed by Stephens with respect to Perkoff's kindred. There are six exceptions in this study. Two are males with nephritis, sons of an apparently healthy mother. Four are normal females who should have inherited the disease from their fathers. The possibility that the four daughters and two mothers concerned may all be unrecognized carriers must be considered. However, one of the normal females (III generation) has borne two normal sons, thus proving that she, at least, does not carry the trait. Haldane's test for incomplete sex-linkage is applied to this kindred as follows:

II Generation Offspring	*Affected*	*Unaffected*
Sex same as that of unaffected grandparent	18	4
Sex different from that of unaffected grandparent	2	15

In spite of the six exceptions listed above, this shows a high probability of incomplete sex-linkage. The total numbers may be compared directly with the 1:1:1:1 ratio which would be expected in the absence of partial sex-linkage:

	D.F.	X^2	*P*
Deviation from 1:1 (affected vs. normal)	1	0.03	> 0.80
Deviation from 1:1 (males vs. females)	1	0.64	> 0.30
Linkage deviation	1	18.69	< 0.0001
	3		

Statistically, then, simple dominance is excluded and partial sex-linkage becomes highly probable. The six exceptions are explained by the crossing-over which occurs in partial sex-linkage. In this case the crossover occurs in every case from the X to the Y chromosome. The appearance of the phenomenon of crossing-over in this disease requires that the defective gene must be on the homologous part of the X-Y chromosome, and statistical analysis led Stephens to make this statement:

> "It would seem from the above data that pyelonephritis in this kindred is inherited as a partially sex-linked dominant trait with 15.4 ± 5.8 per cent crossing-over. This would locate the gene on Haldane's chromosome map 15.4 units below the endpoint of the non-homologous section, in the neighborhood of the loci for xeroderma pigmentosum and Oguchi's disease."

Genetic studies of the deafness aspect are incomplete. Stephen's series is complicated by the introduction of otosclerosis into the kindred at II generation. When this factor is eliminated from consideration in this series, it becomes most likely that the deafness is transmitted as a sex-linked recessive, but this study actually cannot be completed in the absence of audiometric data on three or more generations.

Ocular and renal anomalies have each been reported in two series, concerning a total of eight individuals. Their occurrence is so infrequent as to be unsuitable for genetic evaluation.

The first reports in the literature were English. Perkoff's extensive kindred is traceable to Wales. Sturtz and Burke were able to trace their kindred back six generations to a grandfather born in Ireland in 1753. This led them to suggest that all cases might have a common Anglo-Saxon ancestry. However, reports by Mamou and Sohar of a French-Hebrew and an Iraqui-Hebrew family, respectively, cast doubt on such an hypothesis.

	Kindred		*Clinical*					*Pathological*			*Inheritance*
Author	GENERATIONS	AFFECTED/-TOTAL	ALBUMINURIA	HEMATURIA	PYURIA	DEAFNESS	LETHALITY	MATERIAL	DIAGNOSES	"FOAM CELLS"	
Dickinson	3	12/16	+++	++	+	0	Mild	None			Simple dominant
Pel	3	19/75	+++	++		0	4 ♂ 1 ♀	None			Simple dominant (?)
Kidd	3	8/15	+++	+++		0	♂ Only	None			Sex-linked dominant
Guthrie, Hurst	3	18/31	+++	+++	+	12/31	4 ♂ 1 ♀	None			Sex-linked dominant
Perkoff et al.	5	81/151	++	+	+++	21/151	7 ♂	3 Necropsies 4 Biopsies	Glomerulo- and pyelonephritis	3 Subjects (3/7)	Partially sex-linked dominant
Mamou et Cattan	2	7/14	+++	++	+	1/14	5 ♀ No ♂	None			Simple dominant
Sohar	2	10/?	+++		+	7	2 ♂	None			Dominant
Sturtz et Burke	4	11/?	++	++		5	6 ♂ 1 ♀	None			Sex-linked dominant
Goldbloom et al	2	5/8	++	+	+++	2/8	2 ♂	2 Autopsies	Chronic pyelonephritis	Both subjects	Simple dominant
Reyersbach et al	4	27/77	+++	+++	+	16/77	8 ♂	1 Biopsy 1 Necropsy	Glomerulo- and Interstitialnephritis	1 Subject	Sex-linked dominant
Robin et al	4	17/32	++		+++	11/32	3 ♂ 2 ♀	None			Simple dominant
Goldman et Haberfelde	5	16/31	+++	++	+	0	6 ♂	3 Biopsies 2 Necropsies	Atypical Chronic Glomerulonephritis	1 Subject	Simple dominant

The Genetic Lesion

In 1927, Alport, equating this disease with some form of glomerulonephritis, speculated that the hereditary factor was a decreased resistance to streptococcal infection. Rinkoff and Stern in 1939 suggested that the kidney itself represented a "locus minoris resistentiae" through some hereditary defect, and was thus more susceptible to local infection. Marshall, without reference to this particular disease, published the opinion in 1948 that a high percentage of idiopathic "scars of the renal cortex" could be explained on the basis of congenital renal vascular dysplasia. He cited evidence that this might also be hereditary.

Smith pointed out in 1957 that a disease which exhibits a familial pattern is probably due to one defective gene:

> "Since there is evidence that a gene exerts its influence only through an enzyme, such a disease is probably related to the absence of an enzyme or the production of an abnormal enzyme. In the final analysis, therefore, the pathogenesis of the syndrome under discussion is probably related to some enzymatic defect necessary to the maintenance of the kidney, the auditory nerve, and perhaps the lens."

The Entity

In spite of the paucity of reported cases, and the considerable variation in the disease as reported by various authors, it is possible to make a working definition of hereditary nephritis, and to set down certain criteria for its diagnosis. The material reviewed here has been tabulated on the following page with respect to clinical, pathological, and genetic data. This is done to emphasize the similarity of the reports when subjected to critical analysis. From the material that is now available, it is not possible to sub-classify hereditary nephritis on a clinical or pathological basis. There may be a genetic basis for separating the entity into two groups: simple dominant and partially sex-linked dominant. The presence of "foam cells" in the kidneys of patients from otherwise dissimilar groups lends some support to the hypothesis that all hereditary nephritis stems from a common genetic lesion.

The following criteria are suggested for the diagnosis of hereditary nephritis:

1. Renal disease is manifested by onset, in the first two decades of life, of albuminuria with hematuria or pyuria. Clinical symptoms may be absent. The disease is progressive and lethal in about half of those affected, males more commonly than females.

2. There is a family history of renal disease, and investigation of several generations will reveal an incidence of the disease approaching fifty per cent in the offspring of affected parents.

3. Audiometric testing will show nerve deafness in an appreciable but at present unpredictable number of those afflicted with nephritis.

Editor's Note: References may be obtained by writing the JOURNAL OF THE KANSAS MEDICAL SOCIETY, 315 W. 4th Street, Topeka, Kansas.

C. P. C.

(Continued from page 315)

that a myelophthisic state occurred during the course of the patient's illness. I believe, however, that this subsided rather nicely as the Letterer-Siwe type cells regressed under therapy. With the onset of the patient's terminal illness the bone marrow responded exuberantly to the infectious process. In our opinion it was pneumonia which was the immediate cause of death.

Dr. Delp: This, of course, is a rather uncommon case. Dr. Mantz suggested an intriguing fact in that this viremia acted as a cancerocidal agent, and it does not seem, even though it was fatal, much more dangerous than x-ray and nitrogen mustards.

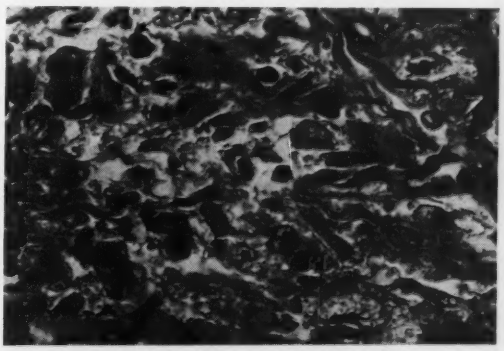


Figure 7. Viral inclusions in abnormal histiocytes in necrotic lymph node involved by Letterer-Siwe's disease.

PATHOLOGICAL ANATOMICAL DIAGNOSIS

Regressing reticuloendotheliosis (histiocytosis X), Letterer-Siwe's type involving mesenteric and retroperitoneal lymph nodes and lymphoid tissue of small and large intestine and probably involving lung, spleen and bone marrow. (History of diagnosis of Letterer-Siwe's disease established by biopsy of skin and iliac lymph node four months before death and of therapy with adrenal corticosteroids and irradiation.)

Diffuse intra-epidermal vesicular dermatitis with intranuclear viral type inclusions of epithelium (varicella).

Focal and confluent fibrinous interstitial pneumonia with varicelliform intranuclear inclusions.

(Continued on page 329)

The President's Message

DEAR DOCTOR:

Our Society secures most of its ideas for changes and improvements from our Committees. It is there that our membership have the opportunity to present their ideas, hear them discussed, and perhaps worked over into resolutions, to be later presented at the Annual Session. Every good active member of the Society who is interested in organized medicine, should want to be a member of a committee, and should have an idea upon which committee he would prefer to serve; he should notify the President-Elect early. At some time during his practice years, he would probably want to serve on the committee representing his specialty, for if he is a member of his Board, he is more likely to be appointed to its Chairmanship. During a physician's active practice years, there is plenty of time for him to serve on several committees successively, and he may serve at the same time on more than one committee.

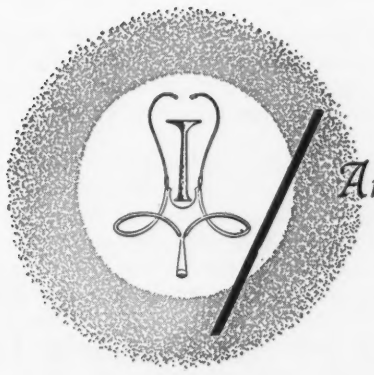
It is usually from the membership of our committees that our Councilors are chosen; lots more time and thought should be devoted to their selection than has usually been the case in the past. Their advice is sought frequently by the Officers of the Society. Nominations for office are often made from the Council, or from those who have served thereon, because of their familiarity with the affairs of the Society, its customs, and mode of operation.

Matters needing consideration and discussion which come to the President's attention, are usually referred to the committee in whose field the matter falls. Thus a tremendous amount of work can be spread about among many committees, and no one person or committee gets into too big an overload of work. Let me urge you all to attend your committee meetings and take an active interest in what is going on in the Society.

Fraternally yours,

Harold M. Glover, M.D.

President



Announcements

Professional meetings, conferences, and postgraduate courses of national importance are listed for the **Doctor's Calendar.** ***Notice of the session is posted in advance to allow the physician time to make preparations.***

Occupational Medicine, a full-time course for physicians, will be given for eight weeks from September 8 through November 10, 1961, by the New York University Post-Graduate Medical School.

The course content covers the following areas: Preventive Medicine, including epidemiology and biostatistics, Administrative Medicine, Occupational Diseases, and Industrial Hygiene. It is aimed at meeting the need for specialized training in industrial medicine. Didactic instruction will be supplemented with field trips to industrial plants, governmental agencies concerned with industrial health and to union health centers. Opportunity will be given to attend medical, surgical, and clinical-pathological conferences held in the New York University Medical Center.

For further information write: Office of the Associate Dean, NYU Post-Graduate Medical School, 550 First Avenue, New York 16, N. Y.

A group of Doctors of the Kansas Medical Society are endeavoring the promotion of a Charter Flight to Europe in June, 1962. The round trip will be from Wichita, Kansas to Paris, France, and will cost approximately $365.00. Those interested please write either Dr. Paul Uhlig, or Dr. John Shellito, 3244 East Douglas, Wichita 8, Kansas. It will be necessary to have a deposit of $100 per seat before the month of September.

The next program for the family physician will be held at the Neurological Hospital, 2625 West Paseo, Kansas City, Missouri, July 30, 1961. The meeting will start at 2:30 p.m.

The theme will be "The Older Patient." Philip B. Reed, M.D., Associate Professor of Psychiatry at Indiana University, Indianapolis, Indiana will be the discussion leader. In the evening after refreshments Dr. G. Wilse Robinson, Jr., will present the "Normal and Abnormal Pathology in the Brain of the Older Patient." M. B. Pettis, M.D. and Abraham Gelperin, M.D. will discuss "Therapies" and "Nursing Management" respectively. General discussion from the audience will follow.

All interested physicians are invited to attend. This program has the approval of the Greater Kansas City Academy of General Practice.

"Cancer Detection" will be the theme of the 15th Rocky Mountain Cancer Conference to be held in Denver, Colorado July 12-13.

This conference is sponsored jointly by the Colorado Division of the American Cancer Society and the Colorado State Medical Society.

Morning sessions will be presented in the form of panel discussions, followed by individual papers by the panel participants. The July 12 panel is "Detect Cancer in Time!—Procedures, Problems and Solutions." The July 13 panel will be on "Neoplasms of the Female Genital Tract."

Further information and program may be obtained by writing Rocky Mountain Cancer Conference, 835 Republic Building, Denver 2, Colorado.

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Editorial COMMENT

Letter to a Young Doctor
Dr. Thomas A. Dooley

Five years ago, Dr. Thomas A. Dooley, 29 years old and just through a hitch in the Navy in Southeast Asia, turned his back on a conventional medical career in the United States and set up a small hospital in the country of Laos, on the border of Red China. To people who had never seen a doctor, or a modern drug, or a hospital, he brought all three. Working with a few like-minded Americans who volunteered their services, Dr. Dooley buried himself in the jungle of northern Laos, emerging now and then to raise more money for more supplies. In spite of bouts with his own illness, he also helped found an international medical assistance organization called MEDICO, which has been establishing and supporting medical centers like Dr. Dooley's in other parts of the world.

Many of us, looking around at the comforts of civilization, the joys of home and family, wonder what makes a man spurn these pleasures for physical discomfort, backbreaking work, the company of misery and the constant challenge of an almost impossible job. We ourselves wondered whether he regretted his choice or whether he would urge it on others. What would he say to a young doctor just starting out? What would he say to all of us who weigh the choices of life?

About six months ago, we asked Dr. Dooley, already ill with cancer, which proved fatal, to write a letter to a young physician which we could publish in THINK. We asked him to consider all the questions we have raised above and to counsel the young doctor on some of the tough problems he faces.

At his hospital in the hills of Laos, Dr. Dooley wrote such a letter for us. We publish it, in this issue, with great pride. We challenge anyone to read this extraordinary testament without being stirred to look searchingly at the values that control his own life.

—THE EDITORS

Young Dr. Dooley (he was only 34 when he died) was one of the world's 10 most admired men, according to a 1960 Gallup poll. He was co-founder of MEDICO (Medical International Cooperation Organization), and a best-selling author. He was the youngest officer in U. S. Medical Corps history to receive the Legion of Merit award, and first American to be decorated by the Kingdom of Laos.

Village of Muong Sing
Kingdom of Laos

Dear Bart,

It is far past midnight. I am sitting at the table in my house at Muong Sing, high in the foothills of the Himalayas in northern Laos. The kerosene pressure lamps overhead are hissing at me, and the wind is lashing down my valley. It whips the palm and frangipani. All the earth on this sad cut of the world seems flooded in the monsoon rains. This is the season of the crashing violence of the tropical storm. The crickets, frogs and wilder jungle animals screech and scream. The high Lao night land is not calm.

But I feel very calm in writing to you. I feel as though I have just met you outside of the medical school auditorium. May I thrust my hand out and say, "Congratulations, Bart. Congratulations on your graduation from medical school. Congratulations on being a doctor." But along with my congratulations, I also want to inject into your mind some thoughts to mull over during your coming year of internship.

Four years of medical school are behind you. "What is past is prologue." You have been given much by parents and teachers. Use it wisely, for others. You have worked hard and learned a good deal, but be-

Editor's Note: This article is reprinted here by permission of MEDICO, Inc. The original copy of these articles appeared in February, 1961, issue of THINK Magazine.

cause you are out of the stress of the doing does not mean that you are yet in the peace of the done. You will never be.

As a doctor, you have a tremendous potential. There are a lot of glorious things ahead of you, if you choose to choose them. I know this very well, though only six years ahead of you in time of practice and age of life.

Know that this passing and precarious time in history will demand much of you. It will maroon the hesitant, but inspire the brave. Stand up and shout, "This is my time and my place in this time!" And seek that place. The state of total gratification is for cows, possibly for birds, not for man. Seek greater things than the material.

Do not aim for just a certain socio-economic position in society. Seek something beyond the split-level ranch house and the two-car garage. Become supremely aware of and intimately involved in the great issues of your day. You have the potential for great deeds, and today demands deeds. Human deeds. Principles enunciated and hopes expressed are not enough. Remember this.

You are a doctor. The proud state of being a doctor is a joyful thing. There is a lot more to you than just the knowledge of bugs and drugs. All the information you have acquired has certainly distilled itself into certain beliefs. Your beliefs may be scattered, rough and unclassified, but you do have them. You are well trained. Your hands are keen, your mind incisive, your sensitivity deep, your vision well honed. You are aware of the sadness of mankind. You know the physiognomy of pain, and, accordingly, the quality of mercy. You know the power of drugs and understand the importance of the "patient who has a sickness" rather than the "sickness that is in the patient." You have a capability to know the pain and glory of other men.

You possess more than knowledge of the healing art. You can do more than laboratory experiments and herniorrhaphies. The greatest attribute of you is that indispensable and essential aspect of your human spirit. Learn how to utilize the fiber and core of your heart.

There is a great deal more to living than just existing. Believing is a fine thing, but placing those beliefs into execution is the real test of strength. The state of being a doctor is a happy one, a lofty one, and one filled with tremendous potential for good. You commit a sin of omission if you do not utilize all the power that is in you. Seek a way to practice your art of medicine, utilizing all the deepest powers of your belief. As a doctor, you must be a part of your time. Isolation and indifference in world affairs are completely past and over. You can no longer be just a doctor or just a researcher or just a teacher. All men belong to me. All men have claims on man. And to the man with special talents, this is a special claim. This is your challenge. It is required that man take part in the actions and passions of this time at the peril of being judged not to have lived at all. You are qualified not only to take part in, but perhaps to lead in the actions of a segment of the world today. A doctor's job is to cure sometimes, to relieve often, and to comfort always.

You must utilize, along with your medical talents, the powers of your spirit and heart. This will help to buttress up the fragile peace of the world.

Bart, you've a year's internship ahead of you. And after that, the choice of a residency for specialty training or private practice. I know you have been plagued with some indecision, "Shall I be a specialist with years and years of more training, or shall I go into private practice now?"

I am going to presume that you will choose the life of a general practitioner. There is a place in the world for specialists (speaketh the young G.P.), but this battered, beaten world of ours needs a few more country doctors in a few more countries and villages, too.

As a general practitioner, where will you practice? There is a need for you every place. But the world is lopsided in its distribution of doctors. Almost all corners of America have available doctors. With veterans' benefits, Social Security, labor union programs, industrial group health plans, and all the others, there is hardly a citizen who cannot find medical attention if he is willing to make some little effort.

I live in Laos. This valley, prior to our Medico hospital, had nothing but black magic, necromancy, witchcraft, clay images, sorcery and betel juice. The villagers wallowed in monkey's blood, cobwebs, tigers' teeth and incantations. They never had hope, much less help. Today, the people of Muong Sing have good medicine, compassionate help, training and a fine little 25-bed hospital. Twentieth century.

You know the world's statistics. The Congo, 13,000,000 people and not one native doctor. South Viet Nam, 11,000,000 people, about 180 doctors. Cambodia, 5,000,000 people, seven doctors. Laos, 3,000,000 people, one Lao doctor. Other nations' statistics are equally staggering.

Though this is sometimes called "the age of the shrug," I do not believe you would say, as some do, "So what, it's not my problem." You know, Bart, you and I are the heirs of all ages. We have the great legacies of music, art, literature and our own medicine. We have been born and raised in freedom. We have justice, law and equality. But we have overlooked the uglier side of our inheritance. We have also the legacy of hatred, bred by careless men before us. We have the legacy of abuse, degradation, and the in-

humanity of men blinded by prejudice, ignorance and personal spleen. To people like you and me, richer in educational opportunities than many, this is a special legacy, and a challenge. To accept the ugly as well as the beautiful and to answer this challenge is a privilege and a responsibility. Accept it without fear.

Bart, I personally believe that the unique aspect of this challenge to young doctors demands that we invest some of our lives in the practice of medicine in foreign fields. I say "some," not a lifetime. This is not expected of us. But we can give a year or two. It can be part of the maturation of a man, the metamorphosis of a doctor.

You went through college, medical school, clerkship; internship lies ahead, maybe residency, and then . . . come to the developing nations of the world for a while. Bring your gadgets, and the armamentarium of drugs, to be sure, but most of all bring your human spirit. Bring your youthful enthusiasm, your drive, your energy, your dedication to help the sick. Bring your wonderful spontaneity, your belief in the good and the right. Bring along a sense of humor, don't forget it; you'll certainly need it when the roof leaks, the patients eat all the pills the first dosage, and the witch doctors put cow dung over your sterile compresses. Bring also a few cents' worth of the spirit of adventure that our founding fathers possessed. Spend some time in valleys like Muong Sing. Invest some of your life to answer personally the challenge of today, your legacy, your heritage.

It is more difficult for other professions to enter and work in a foreign country. Citizens and governments are suspicious . . . as they are even a bit suspicious of the doctor. (There are some who are not yet convinced that I am not an agent of the FBI or a Jesuit-in-disguise.) But your M.D. diploma does open many doors. You will be allowed to come to these underdeveloped areas and build your small hospital. You can cure sometimes, relieve often, and comfort always. Splash some of the warmth and goodness of your human spirit on people who heretofore knew little of this element in the Western man. My villagers' lives were just one great groan of agony before the Medico hospital was built. They knew only Western men bent on colonizing them, and perhaps exploiting what little they had.

Your medicine will have a twofold effect. You will find that by just being a doctor with qualities of the human heart, you will help to unify men. Simple humanity makes the primitive lands of Asia and Africa important to every American. Simple self-interest makes it vital.

You are probably thinking, "Tom, hate to sound this way, but what's in it for me? We are all a little selfish, you know." Right you are, Bart. Perhaps we should be a bit wary of the man who is completely unselfish. There is a great deal "in it" for you. By investing a portion of your life for work here, by depositing a year or two of your time you will take back with you into private practice a great sense of accomplishment, coupled with a vast human experience. Your accomplishments will be on a wider scope; along the broad horizons of peace for the whole world. You will always know that you have given a fragment of your life for the good of many.

All men yearn to lose themselves in something greater than themselves. You will have done this, and will have helped to achieve that unquenchable promise that someday all men of all races will learn to live together in peace. I do not believe this fulfillment is achievable in private practice.

You've always been a bit of a cynic. I imagine you are quietly snarling, "Okay, Tom, you've made your point, but to give up a year or two for a spiritual thing called 'fulfillment,' don't know 'bout that."

I can only remind you that the history of mankind constantly repeats the exclamation that the only way man can achieve his own happiness is to strive for the happiness of others. And you reply, "Ugh, Dooley's murky mysticism again." History also proves, Bart, that men rarely learn from the teachings of history, but must learn for themselves. Come to Asia, Learn.

There are programs in the world with which you could work. Programs like the World Health Organization, our own Medico, and others. These will pay you enough to keep you out of debt (though perhaps not much more). They will handle the mechanics of medical procurement and supply. Medico, you know, from a kernel of an idea in 1958, now has 17 hospitals, clinics and programs in 12 different nations. Dozens and dozens of doctors like you. Free next year, Bart?

Today demands a deeper emphasis on the brotherhood of man. All professions must seek ways to do this. For the doctor it is not a difficult thing. It is in the root of the tree of a doctor to understand and believe in brotherhood. This concept was not so important in the times of our fathers. It is now. The Brotherhood of Man exists as definitely as does the Fatherhood of God. And we must not forget it.

I do not believe "brotherhood" is a sentimental, mushy-mouthed hyperglycemic thing. It is a potent, mighty force to bring men together as men. We are not as actively engaged in solidifying this idea as we should be. Doctors know the alikeness of all men. The world does not need another union of white men, or of American men, or of Dutch men, or of Negro men. Or more fragmentation of peoples into endless exclusivisms. Brotherhood should be a force to unite men . . . as men.

Patriotism is not enough, either. Nations must belong to a larger world, with a wider horizon than that

of any single country. Countries working together, each giving something to the other . . . this is part of brotherhood. Asia and Africa need picks and shovels, bulldozers and syringes to remove the high cliffs of poverty, injustice and sickness. You as a doctor have those syringes. We just do what we can, as individuals, to help other individuals.

We young Americans must take the drama of our freedoms (from diseases as well as from tyranny) that we have received from the past and project it into the future. For other men. We who have it must help those who do not have it.

The kerosene is running out of the lamps and they the sputtering and flickering. Dooley needs to hit the sack, so I'll stop the letter just now . . . and continue it tomorrow.

A full day has passed since I began this letter to you. At the clinic this morning we had 78 patients. Everything from a blazing malaria to a man who brought his donkey, requesting that we suture up a laceration in its flank. We gave the malaria patient chloroquine, and sutured the laceration in the donkey's posterior with chromic catgut, size 2 (tough ass).

Some children had diarrheas, eye inflammations, and one had a case of head lice. My American corpsmen pulled some teeth (dentistry is not for me). The kids howled just like they do in America. The old gals complained about having to wait in line, just like they do in America. A few of the older gents wanted some "vigor pills," just like . . . well, anyway. There are no really deep differences between people. I have spent six years of my life among different men, and always I find the similarities outweigh the differences. Each life is infinitely precious as a life. Everywhere.

To recapitulate what I've written, Bart, I believe that as a young doctor, as soon as you finish your internship, you should spend a year or two in lands such as Laos. You should utilize your profession and your heart as a cable to bind men together. Danger cements men; why can't other forces be used? Many tools must be implemented to destroy the false walls that separate us. Medicine is one. Medicine when enveloped in that indispensable element of the human spirit. Kindness and gentleness are daily instruments of the doctor, more than of other professions. Kindness and gentleness can be potent weapons to fight against the anger of the world.

The world is made up of persons. Internationality is only a conglomeration of individuals. All individuals yearn for something human. This flings a special challenge to you as there is no more intimate person-to-person relationship than that of the doctor and his patient. Bring the talents of your degree, and the spirituality of your heart, to distant valleys like mine. And take back with you a rich, rich reward.

Dedicate some of your life to others. Your dedication will not be a sacrifice. It will be an exhilarating experience because it is intense effort applied towards a meaningful end.

So along with my congratulations on your graduation I send my wish that you will utilize yourself as a force of unity in the fragile peace of today. And that you will know the happiness that comes of serving others who have nothing.

Sincere best wishes always,

TOM

C. P. C.

(Continued from page 323)

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It was the vigorous pronouncements Jesus made on controversial matters that sent Him to the cross. If He had confined Himself to little Mickey Mouse morals, He would never have been heard of.—*Dr. Edwin T. Dahlberg*

Those who cannot miss an opportunity of saying a good thing are not to be trusted with the management of any great question.—*William Hazlitt*

When you have found out the prevailing passion of any man, remember never to trust him where that passion is concerned.—*Lord Chesterfield*

. . . no man is entirely unmoved by being asked for information upon his particular subject. . . .

—*Lord Dunsany*

In skating over thin ice our safety is in our speed. —*Ralph Waldo Emerson*

The Kansas Press Looks at Medicine

Editor's Note. In this section the JOURNAL reproduces editorials relating to medicine which have appeared in the lay press. An effort is made to include both favorable and unfavorable comments, and the Editorial Board in no instance assumes responsibility for the opinions expressed.

SOURCE OF DOCTOR SUPPLY

A while back it was discovered that some of the foreign physicians seeking internships and residencies in American hospitals were below the standard set for American medicine. A more rigid screening program was instituted. On April 4 examinations were given at 132 centers in the United States and abroad.

Results of these tests happily promise a good increase in the number of physicians and internes available in this country. Of the 5,326 who took the examination, 2,063 got grades high enough for full qualification and another 1,646 are provisionally qualified and will be permitted to study until they have increased their knowledge and techniques.

This is double the number of qualified foreign doctors found in try-outs a year ago and gives promise that there will be a steadily increasing source of supply abroad. It does not cancel the necessity of more medical training of Americans to increase the doctor supply enough to keep pace with population. But it helps.—*Wichita Eagle,* May 27, 1961.

DOCTORS FACE AN ISSUE

The proposal to finance health care for the elderly through higher social security taxes waxes hot and heavy. The Kennedy administration supports the measure while the American Medical Association opposes it.

It has been mentioned before that doctors, including your family doctor, haven't depended on the printed word to any great extent as a public relations outlet. There has been no important reason for medical doctors to come to their own defense.

On the other hand, politicians have never avoided the printed word. In fact they offer publications a huge supply of publicity. When the chips are down the politician is out in front with his public relations and know-how. The medical doctors are feeling for the first time the stinging scratches that words can make on their tender hides. Even Walter Reuther's union is submitting handouts to the press favoring the health program.

Slowly the doctor is toughening-up to the attack. He's building new defenses and in time will take the offensive. It's difficult to tell a physician he should be in the public eye. This time he has to be to save his own hide from socialism.

One move in the right direction came from the Jefferson County (Ohio) Medical Society when it tossed a terse reminder to the public by way of an advertisement in the Steubenville *Herald,* a newspaper of 30,000 circulation.

The doctors did not use the advertisement to argue the issue. Instead they bluntly stated in big type: "We know of no one who has been denied medical care because they could not afford it." Then came an offer to reply to that claim. "Any expression of opinion is welcome," the ad said and gave a box number to write.

After a month the medical society had received two replies. One letter complained about the length of time that had to be spent in a relief office. The other was from a man whose wife was hospitalized at the time he wrote.

It would appear that if the health of the elderly is in such a horrible state there would have been thousands of letters stating cases. These would come not only from the elderly, but from members of the family and citizens who were generally concerned.

Like so many proposals that stem from the political brain center, this health plan is supposed to have appeal if the public can be persuaded to generate

(Continued on page 347)

Official Proceedings

Conclusion of the report of 1961 meeting of the House of Delegates

The transactions of the 102nd Annual Session were published in the June issue of THE JOURNAL and will be concluded here.

Included are all resolutions in numerical order as they were adopted by the House of Delegates. Eliminated from the minutes as preserved in the executive office are such things as who made the motions, the discussion, amendments, and the many formalities of the meeting. Since these do not reflect Society policy, the proceedings will list only the resolutions in the form in which they were adopted.

In several instances two or more resolutions were applied to similar subjects. These were combined by reference committees into one. A few resolutions were not adopted. Those are not recorded in this report.

RESOLUTION NO. 32

General Practice Awards

Be It Resolved: That subject to the approval of the House of Delegates of the Kansas Medical Society and the membership of the County Medical Societies of the State of Kansas, an Award be established known as "The Practitioner of the Year Award," and that same Award be presented to one or more outstanding physicians each year. The means and basic qualifications necessary for selecting such nominees for this Award to be determined in the future.

RESOLUTION NO. 33

WHEREAS, in this nation it has always been the responsibility of the individual to provide for his own health care, and

WHEREAS, this is held to be a privilege, and

WHEREAS, the cost of such care at times presents an economic crisis to the individual, and

WHEREAS, the Congress of the United States, recognizing the above, passed the Kerr-Mills Bill which will provide federal grants to states wishing to aid persons over 65 years of age who lack the resources to pay for their health services, and

WHEREAS, this is in keeping with the tradition upon which this nation was founded,

Therefore Be It Recommended that the State of Kansas create a program whereby benefits under the Kerr-Mills Bill may become available to the eligible citizens of this state.

RESOLUTION NO. 34

Committee on Gerontology

WHEREAS, the best argument against Federal intervention in health care for the aged is in the proof that such intervention is unnecessary, and

WHEREAS, there has not been an adequate compilation of services and facilities available to persons of 65 years of age of Kansas, therefore

Be It Resolved that the Committee on Gerontology be authorized to proceed with a survey to determine the availability of health care to persons of 65 years of age and over, and

Be It Further Resolved that other statewide organizations interested in the subject be invited to participate in this survey, and

Be It Further Resolved that when the information is obtained, a full report will be made to the Council which shall determine if the information should be published.

RESOLUTION NO. 35

Committee on History

Be It Resolved that the membership cooperate in the various community celebrations of the Kansas Centennial in such a manner as they may approve such as, floats, pageants and speeches that depict the pioneer doctor of medicine.

RESOLUTION NO. 36

Committee on History

Be It Resolved that the book *The Kansas Doctor* be placed in as many libraries in Kansas as possible and if possible to be in memory of some of the older physicians of that community.

RESOLUTION NO. 37

Committee on History

Be It Resolved that an effort be made to stimulate interest in the medical sudents at the University of Kansas in the "History of Kansas Medicine" by offering an annual award to the students submitting papers on that subject if the paper dealt with a Kansas physician or a member of the University of Kansas School of Medicine faculty and that these

papers be placed in a special file in our central office —this award to be in addition to the Guffey Awards that are now given for this subject, and that the Council be directed to make a final decision on this subject.

RESOLUTION NO. 38

Committee on Hospitals

WHEREAS, two (2) years ago the House of Delegates approved the formation of the Kansas Voluntary Advisory Commission to establish professional standards for the small hospitals of this state, and

WHEREAS, this project has been approved by the Kansas Hospital Association, and

WHEREAS, the Kansas Hospital Association wishes to add to these a set of standards for services other than professional which have not yet been accomplished, therefore

Be It Resolved that the Committee on Hospitals be authorized to continue negotiations with the Kansas Hospital Association in an effort to complete this project, and

Be It Further Resolved that the standards for professional services as previously approved by this Society be now submitted to the hospitals of Kansas and to the chief of staff of each hospital in the state with the invitation that they voluntarily subscribe to these principles at this time.

RESOLUTION NO. 39

Committee on Industrial Medicine

WHEREAS, the Commissioner of Workmen's Compensation in Kansas has requested Kansas Medical Society cooperation in incorporating the current Relative Value Studies into a revised Workmen's Compensation Manual, and

WHEREAS, the Committee on Industrial Medicine has gone on record as favoring a $5.00 point value to the Kansas Relative Value Studies for use in Workmen's Compensation cases

Be It Resolved that the Kansas Medical Society endorse the revised 1961 Kansas Relative Value Studies for use in the new Workmen's Compensation Manual with a point value of $5.00 as the factor for determining the fees in compensation cases, and

Be It Further Resolved that the Society Committee on Industrial Medicine and the Executive Staff be authorize to assist and cooperate with the Commissioner in every possible way.

RESOLUTION NO. 40

Committee on Medical Economics

WHEREAS, the Kansas Medical Society Committee on Medical Economics is charged with the responsibility of overseeing all phases of the Society's various group insurance programs, and

WHEREAS, there is at all times the possibility of a misunderstanding between an individual member of the Society and an insuring company or soliciting agency, and

WHEREAS, the Society desires to protect the interests of any member covered under a Society sponsored insurance program

Be It Resolved that the Kansas Medical Society endorse the requirements that any difficulty between a member of the Society and an insuring company or soliciting agency be reported to the Executive Office by the company or agency involved in order that the difficulty may be referred to the Medical Economics Committee and the Executive Committee for consideration.

RESOLUTION NO. 41

Committee on Medicare

WHEREAS, 95 per cent of persons eligible under the Medicare program have an annual income of $3,000 or less, and

WHEREAS, the National Medicare Program is founded on the premise that professional services shall be reimbursed at usual charges for persons of low income, and

WHEREAS, the Kansas Medicare contract comes up for re-negotiation in July of this year, and

WHEREAS, Medicare pays the expenses for three (3) persons to Washington for the purpose of negotiating such a contract

Be It Resolved that the president and whoever he designates to accompany him, together with one representative from Kansas Blue Shield, be authorized to negotiate this contract, and

Be It Further Resolved that the fee schedule shall be determined by the Kansas Relative Value Studies with a point value approximating Blue Shield *Plan B.*

RESOLUTION NO. 42

Membership Orientation

WHEREAS, new physicians in Kansas often lack information concerning health resources, health regulations and services available to the physicians through the Kansas Medical Society, and

WHEREAS, this committee has assembled material on this subject

Be It Resolved that packets of such information be distributed to each councilor with the recommendation that he give one to each new physician as he enters practice in the individual councilor's district, and

Be It Further Resolved that such material shall be assembled at little or no cost to the Kansas Medical Society through the utilization of existing printed documents, to be supplemented by mimeographed material prepared by the Committee.

RESOLUTION NO. 43

Committee on Mental Health

WHEREAS, Senate Bill 212, as passed by the 1961 Kansas Legislature places under the Board of Social Welfare the supervision of all mental health activities of this state, and

WHEREAS, House Bill 125, authorizes communities to create mental health clinics, therefore

Be It Resolved that Committee on Mental Health be authorized to work closely with the Division of Institutional Management of the State Board of Social Welfare and with the Kansas Mental Hygiene Society in the organization and operation of such clinics, and

Be It Further Resolved that Committee on Rural Health be directed to inform the component societies of this state of its recommendations in the establishment of such clinics, and

Be It Further Resolved that the Committee on Mental Health be directed to give assistance to component medical societies wishing to implement such clinics.

RESOLUTION NO. 44

Committee on Mental Health

WHEREAS, the mental health program of Kansas has been placed under the direction of State Board of Social Welfare

Be It Resolved that the Committee on Mental Health be authorized to work with the Division of Institutional Management in the preparation of a commitment act to be submitted to the 1963 Kansas Legislature.

RESOLUTION NO. 45

Committee on Pathology

WHEREAS, the Committee on Pathology and other interested physicians attempted to pass a revision of the Coroner's Law, and

WHEREAS, the reasons this failed to pass now appear to be understood, therefore

Be It Resolved that the Committee on Pathology be authorized to prepare a revised Coroner Bill, and

Be It Further Resolved that this Committee, together with other members of the Kansas Medical Society, shall meet with the following organizations as often as may be necessary to obtain their endorsement: the Kansas Official Council, the Kansas Funeral Directors Association, and the Kansas Bar Association, and

Be It Further Resolved that the Committee on Pathology be authorized to meet with individuals who might have a special interest in this subject, and

Be It Further Resolved that when such proposed legislation has been prepared to be submitted to the Council and that when the Council so approves, each component medical society be provided with this material and be requested to enlist the support of interested local organizations in visiting with members of the Kansas Legislature prior to the opening of the 1963 Session.

RESOLUTION NO. 46

Committee on Post Graduate Study

WHEREAS, the graduate education programs provided through the University of Kansas School of Medicine are increasing in popularity, and

WHEREAS, the Committee on Post Graduate Education has as its primary function the stimulation of such effort

Be It Resolved that the Dean of the School of Medicine be advised this Committee will assist him in the planning and preparation of such programs, and

Be It Further Resolved that the importance of this work makes it appear advisable that at least two (2) meetings be held annually with the Medical School, one of which would be in the fall and one in the spring.

RESOLUTION NO. 47

Public Policy

WHEREAS, the American Medical Association has established "Guides for Conduct of Physicians in Relationships with Institutions," adopted December, 1951, and reaffirmed December, 1959, and

WHEREAS, the American Medical Association has established "Principles of Medical Ethics" as adopted June, 1957, and

WHEREAS, it is a matter of concern to the Kansas Medical Society that violations of these principles occur, and

WHEREAS, the Kansas State Board of Healing Arts has recently stated that, "Hospitals in Kansas are not authorized to engage in the practice of Healing Arts as defined by the Kansas Healing Arts Act, and that all professional services performed in the practice of the Healing Arts must be performed by or under the supervision of a duly licensed practitioner," and

WHEREAS, the practice of medicine in any of its specialties by any hospital, corporation or lay body, whether for profit or non-profit, is undesirable, therefore

Be It Resolved, that the Kansas Medical Society endorses these actions and opinions and that it shall be the policy of the Kansas Medical Society that, "a physician should not dispose of his services under terms or conditions which tend to interfere or impair the free and complete exercise of his medical judgment and skill or tend to cause a deterioration of the quality of medical care," and that "a physician should not dispose of his professional attainments or services to any hospital, corporation or lay body by whatever name called or however organized under terms or conditions which permit the sale of the services of that physician by such agency for a fee," and

Be It Further Resolved, that these resolutions do not refer to remuneration of a physician for teaching, research, charitable service, administration and supervision of health services of a corporation, administration or government service. A corporation or lay agency properly may provide such services and employ or otherwise engage physicians for these purposes, and

Be It Further Resolved, that professional services shall, and hospital services shall not, include the practice of pathology, radiology, anesthesiology, physical medicine, or any other medical specialty as defined by law and the Kansas Medical Society, and

Be It Further Resolved, that services performed by a physician and the technical staff under his supervision shall be recognized as professional service and identified to the patient as such. The fees established for professional service and collection of said fees are the responsibility of the physician; however, if mutually agreeable, the hospital and/or clinic may act as collecting agent. The amount paid to or otherwise retained by the hospital for space, equipment, collections, or other expense related to the department used by the physician(s) is a matter to be agreed upon by the physician and the hospital.

RESOLUTION NO. 48

School Health Committee

WHEREAS, the American Medical Association Committee on school health, the National Education Association Committee on school health, the American Academy of Pediatrics Committee on school health, and other authoritative organizations have recommended that interscholastic competition in junior high schools and elementary schools be discontinued. The School Health Committee of the Kansas Medical Society recommends that the Kansas Medical Society adopt a policy recommending the abandonment of interscholastic competition at the junior high and elementary level.

RESOLUTION NO. 49

Committee on State Meeting Format

WHEREAS, the State Meeting Format Committee met in Salina during the Summer of 1960, to plan meetings for the next two (2) years

Be It Resolved that the 1962 Annual Session be held in Kansas City, Kansas, April 30-May 2, and that the general schedule of the 1961 annual meeting be followed for 1962, and

Be It Further Resolved that the Wyandotte County Committee explore the possibility of the use of live television for a portion of the scientific meeting, and

Be It Further Resolved that the 1963 Annual Session be held in Salina, May 6-8.

RESOLUTION NO. 50

Special Committee on Diabetes

WHEREAS, this committee was appointed for the first time this year and the following recommendations represent new Society policy

Be It Resolved that the medical profession in local areas of Kansas unite with allied professional groups in their area to form local Diabetes Associations and when such organization is completed, lay persons from the community be invited to participate with them, and

Be It Further Resolved that local Diabetes Associations should be urged to participate in group discussions directed toward public information but that no effort at treatment should ever be considered by such associations, and

Be It Further Resolved that a letter be sent by this Committee to the secretaries of each component society in Kansas recommending their participation in the organization of a local Diabetes Association and offering, if they desire, the services of the Diabetes Committee in assisting them with the details of organization.

RESOLUTION NO. 51

Special Committee on Diabetes

WHEREAS, the detecting of the presence of Diabetes is the practice of Medicine

Be It Resolved that diabetes detection drives in any community shall be approved by the local medical society and medical leadership shall be provided by the society before such drive is started, and

Be It Further Resolved that the Committee on Diabetes be authorized to recommend to each county medical society the establishment of periodic local detection services.

RESOLUTION NO. 52

Special Committee on Kansas Health Facilities Information Service

WHEREAS, the Kansas Medical Society has cooperated with the Kansas Hospital Association, and

WHEREAS, Dr. F. E. Wrightman, president, and Dr. Glenn R. Peters, immediate past president, and the Executive Secretary have represented the Medical Society in this project, and

WHEREAS, there is a report mimeographed on this subject in the hands of the members of the House of Delegates

Be It Resolved that the Kansas Medical Society become a charter participant in the incorporation of the Kansas Health Facilities Information Service, Inc., and

Be It Further Resolved that the substance of the By-Laws as recommended be approved, and

Be It Further Resolved that the Council select the Kansas Medical Society voting member to this organization, and

Be It Further Resolved that the Council be authorized to decide whether a financial contribution shall be made to this organization which, if decided in the affirmative, shall not be more than $500.00.

RESOLUTION NO. 53

Special Committee on Treatment of Narcotic Addicts

WHEREAS, the treatment of narocotic addicts by confinement in a drug-free hospital under compulsory civil commitment may be the preferable present method of treating narcotic addiction, but

WHEREAS, if this is the only authorized mode of treatment it would preclude research and exploration that might lead to the discovery of new and more effective treatment, and

WHEREAS, it is well recognized that no present method of treating the narcotic addict is completely satisfactory, therefore

Be It Resolved that the Kansas Medical Society generally endorses hospital confinement for the narcotic addict and recommends that additional federally operated hospitals be constructed for this service as may be needed, and

Be It Further Resolved that penalties, especially those for the unlawful selling of narcotics, be greatly increased, and

Be It Further Resolved that the treatment of a narcotic addict is an individual problem as is the medical treatment of any other condition and that no arbitrary statement of policy endorsing only one mode of treatment to the exclusion of all others can be accepted as a sound medical policy, and

Be It Further Resolved that continued research be conducted to discover more effective methods of treatment which in this instance is especially important because current methods of treatment are notoriously inadequate, and

Be It Further Resolved that every possible opportunity for rehabilitation of the narcotic addict be made available to him, and

Be It Further Resolved that the Kansas Medical Society assume leadership in this state for increasing education on this subject especially among the professional schools and in the societies of Medicine, Dentistry, Nursing and Pharmacy, and

Be It Further Resolved that this resolution be introduced by its Kansas Delegates at the House of Delegates of the American Medical Association.

RESOLUTION NO. 54

Special Committee on Podiatry

WHEREAS, the podiatrists of Kansas are examined and receive their license from a board consisting of a licensed podiatrist and two licensed doctors of medicine, one of which shall be the secretary of the Healing Arts Board, and

WHEREAS, a Kansas license in podiatry requires a minimum of one (1) year college and four (4) years education in an approved school of podiatry which is affiliated with one approved school of medicine, and

WHEREAS, a podiatrist is permitted to treat conditions below the ankle except that "no podiatrist shall amputate the human foot or toes, neither shall any podiatrist administer any anesthetic other than local . . . ," and

WHEREAS, in such practice the podiatrist is authorized the use of all drugs including narcotics, if the treatment is for a condition occurring below the ankle, and

WHEREAS, the American Medical Association through its Judicial Council has ruled podiatry to be not a cult and that doctors of medicine may ethically consult with podiatrists and teach in their schools, and

WHEREAS, some items of care legally given by podiatrists are listed among benefits for which Kansas Blue Shield members are entitled to coverage, and

WHEREAS, the Blue Shield plans in some states pay such services to podiatrists and at least one national contract in which Kansas participates, the coverage for Federal employees, includes payment for podiatrists' services, and

WHEREAS, it is believed some of the above information may not have been known when the Blue Shield Board and the House of Delegates acted upon this question, therefore

Be It Resolved that the Council consider the question in the light of the above information and, if agreeable, re-submit the proposition to the next session of the House of Delegates, and

Be It Further Resolved that the House of Delegates vote upon the proposition to authorize the Kansas Blue Shield Board to receive from the podiatrists of Kansas a list of procedures they wish to perform for Blue Shield subscribers and that the Blue Shield Board then determine, without opposition from the Kansas Medical Society, which, if any, procedures can comply with the benefits in the subscribers' contract and within the practice privileges authorized under the podiatrists' Kansas license, and

Be It Further Resolved that the Blue Shield Board then determine, without opposition from the Kansas Medical Society, which, if any, of the procedures so determined shall be paid when performed by podiatrists.

RESOLUTION NO. 55

WHEREAS, the duties of the Executive Staff Officer of the Kansas Medical Society have greatly increased in scope and importance as a state and national position representing the organized medical profession of the State of Kansas, and

WHEREAS, there is a growing trend in major medical organizations to use such titles as Executive Director, etc., for their chief executive staff officer, and

WHEREAS, the duties of the Kansas Medical Society Executive Staff Officer, as described in the By-Laws of the Society, Chapter VII, Section 7, are those of an administrative officer in charge of directing and discharging the charges of the Society, and

WHEREAS, his duties, while encompassing those of keeping the records and minutes of the Society are carried out in an administrative way and the physical preparation of these is done by other persons, therefore

Be It Resolved that the title of the executive staff officer of the Kansas Medical Society be changed to the Executive Director to denote the dignity of this office, and

Be It Further Resolved that the Constitution and Rules Committee be instructed to make the necessary revision in the By-Laws.

RESOLUTION NO. 56

WHEREAS, organized medicine has been condemned as being against all medical legislation, and

WHEREAS, the Kerr-Mills Bill has been endorsed by the Kansas Medical Society, and

WHEREAS, no publicity has been given to this action, therefore

Be It Resolved that the public be acquainted with the stand taken by the Kansas Medical Society by all means of publicity available.

RESOLUTION NO. 57

WHEREAS, in 1959, there was introduced in the House of Delegates Resolution No. 31, calling for the establishment of a Commission to Study the Relation of Medicine to Optometry and to report to the House of Delegates, and

WHEREAS, the House of Delegates caused to be established a subcommittee to study the relation of medicine to optometry, under the then Joint Committee to Study Paramedical Areas in Relation to Medicine, and

WHEREAS, the original Joint Committee to Study Paramedical Areas in Relation to Medicine has been succeeded by the Committee of Relationship of Medicine With Allied Health Professions and Services, and

WHEREAS, optometrists are not ancillary to Medicine, but are independent licensed practitioners and, therefore, do not constitute an allied health profession, and

WHEREAS, there exists confusion in the public mind as to the distinction between medical care for patients with ocular complaints and optometric services, and

WHEREAS, the lack of understanding in this area is a threat to the welfare of the patient, therefore

Be It Resolved that the House of Delegates establish a Commission on the Relation of Medicine to Optometry, to be appointed by the Speaker of the House at least one-half the members of which Commission shall be physicians practicing in the ophthalmological branch of Medicine, and

Be It Further Resolved that it shall be the specific function of this Commission to conduct a broad study, from the standpoint of the public interest of the problems involved in the present relation of Medicine to Optometry, and to explore all possible and desirable solutions to these problems, and

Be It Further Resolved that the Board of Trustees be requested to provide adequate personnel and funds for the proper performance of the duty assigned to this Commission, and

Be It Further Resolved that this Commission shall report to the House of Delegates not later than June, 1962.

RESOLUTION NO. 58

WHEREAS, many general practice residencies are unfilled and many have inadequate progressive and integrated training programs, and

WHEREAS, the present two-year family practice pilot program of the American Medical Association

(Continued on page 346)

Committees for 1961-1962

Allied Groups

J. B. Pretz, Kansas City, Chairman; J. J. Basham, Fort Scott; M. L. Belot, Jr., Lawrence; H. O. Bullock, Independence; A. R. Chambers, Iola; W. M. Cole, Wellington; G. W. Cramer, Parsons; R. A. Crawford, Hutchinson; F. J. Eckdall, Emporia; F. B. Emery, Concordia; J. H. Gilbert, Seneca; R. M. Glover, Newton; D. A. Huebert, Wichita; K. A. Powell, Leavenworth; D. J. Smith, Overland Park; N. C. Smith, Arkansas City; M. O. Steffen, Great Bend.

Anesthesiology

R. T. Parmley, Wichita, Chairman; H. R. Barnes, Hutchinson; H. J. Brown, Winfield; W. E. Enders, Kansas City; E. L. Frederickson, Kansas City; V. G. Henry, Newton; M. R. Knapp, Wichita; R. S. McKee, Leavenworth; W. O. Martin, Topeka; A. W. Mee, Wichita; W. F. Powers, Wichita; L. J. Ruzicka, Concordia; H. F. Spencer, Emporia; J. R. Sumner, Hutchinson; E. T. Wulff, Atchison.

Auxiliary

C. H. Benage, Pittsburg, Chairman; W. T. Braun, Pittsburg; H. L. Barry, Wichita; V. E. Brown, Sabetha; R. H. O'Neil, Topeka; L. G. Glenn, Protection; E. B. Scagnelli, Dodge City; L. H. Leger, Kansas City; J. B. Jarrott, Hutchinson.

Blue Shield Relations

H. R. Schmidt, Newton, Chairman; P. L. Beiderwell, Belleville; F. J. Bice, WaKeeney; C. W. Bowen, Topeka; R. E. Capsey, Centralia; O. R. Cram, Larned; C. W. Erickson, Pittsburg; G. W. Fields, Scott City; L. G. Glenn, Protection; A. C. Hatcher, Wellington; P. E. Hiebert, Kansas City; H. P. Jones, Lawrence; W. R. Lentz, Seneca; K. L. Lohmeyer, Emporia; J. R. Neuenschwander, Hoxie; B. G. Smith, Arkansas City; E. A. Walsh, Onaga.

Child Welfare

R. L. Dreher, Salina, Chairman; M. J. Blood, Wichita; R. D. Boles, Dodge City; M. S. Boyden, Lawrence; W. H. Crouch, Topeka; D. R. Davis, Emporia; F. A. Gans, Salina; T. C. Hurst, Wichita; A. C. Irby, Fort Scott; G. F. Jordan, Jr., Wichita; H. P. Jubelt, Manhattan; W. F. McGuire, Wichita; E. A. Martin, Parsons; E. T. Olson, Newton; P. T. Schloesser, Topeka; R. N. Shears, Hutchinson; C. J. Winter, Jr., Kansas City; T. E. Young, Topeka.

Conservation of Eyesight

M. S. Lake, Salina, Chairman; B. J. Ashley, Topeka; E. J. Bribach, Atchison; L. L. Calkins, Kansas City; M. A. Carter, Wichita; C. A. Crockett, Kansas City; J. E. Hill, Arkansas City; D. O. Howard, Wichita; C. T. McCoy, Hutchinson; H. E. Morgan, Newton; H. L. Patterson, Larned; W. M. Scales, Hutchinson; E. T. Siler, Hays; D. P. Trimble, Emporia.

Conservation of Hearing and Speech

J. A. Budetti, Wichita, Chairman; C. W. Armstrong, Salina; R. E. Bridwell, Topeka; H. R. Draemel, Salina; E. K. Enns, Newton; E. S. Gendel, Topeka; C. L. Gray, Wichita; W. P. McKnight, Wichita; E. E. Miller, Pittsburg; R. G. Montgomery-Short, Halstead; V. R. Moorman, Hutchinson; W. D. Pitman, Pratt; G. O. Proud, Kansas City; R. E. Riederer, Olathe.

Constitution and Rules

A. W. Fegtly, Wichita, Chairman; T. C. Ensey, Marion; A. C. Harms, Jr., Kansas City; L. S. Nelson, Jr., Salina; A. S. Reece, Gardner; G. L. Thorpe, Wichita; M. M. Tinterow, Wichita; C. E. Vestle, Humboldt; E. D. Yoder, Denton.

Control of Cancer

C. R. Openshaw, Hutchinson, Chairman; J. P. Berger, Wichita; G. L. Campbell, Arkansas City; W. G. Cauble, Wichita; A. M. Cherner, Hays; A. G. Dietrich, Newton; J. C. Dysart, Sterling; L. S. Fent, Newton; A. A. Fink, Topeka; W. A. Grosjean, Winfield; W. J. Kiser, Wichita; J. R. Kline, Wichita; M. V. Laing, Kansas City; C. H. Miller, Parsons; N. C. Nash, Wichita; D. C. Reed, Wichita; L. W. Reynolds, Hays; R. H. Riedel, Topeka; D. S. Ruhe, Kansas City; P. H. Schraer, Concordia; B. E. Stofer, Wichita; L. E. Vin Zant, Wichita; J. W. Welch, Halstead; H. M. Wiley, Garden City.

Control of Tuberculosis

P. R. Carpenter, Kansas City, Chairman; A. L. Ashmore, Wichita; E. A. Baude, Topeka; R. M. Brooker, Topeka; J. A. Butin, Chanute; R. I. Canuteson, Lawrence; L. H. Coale, Kansas City; R. F. Conard, Emporia; M. J. FitzPatrick, Kansas City; J. K. Fulton, Wichita; E. C. Hwa, Newton; J. L. Morgan, Emporia; J. M. Mott, Topeka; G. W. Nice, Topeka; C. Pokorny, Halstead; C. F. Taylor, Norton; F. A. Trump, Ottawa; P. H. Wedin, Wichita.

Diabetes

B. M. Matassarin, Wichita, Chairman; M. L. Bauman, Wichita; R. E. Bolinger, Kansas City; J. E. Crary, Topeka; N. M. Jenkins, Salina; T. J. Luellen, Wichita.

Emergency Medical Care

H. H. Hyndman, Wichita, Chairman; G. L. Ashley, Chanute; K. F. Bascom, Manhattan; F. C. Beelman, Topeka; R. W. Blackburn, Council Grove; G. L. Gill, Sterling; A. J. Horejsi, Ellsworth; C. C. Hunnicutt, Sabetha; N. M. Jenkins, Salina; D. S. Klassen, Newton; J. T. Mott, Topeka; N. H. Overholser, El Dorado; G. E. Paine, Hutchinson; O. F. Prochazka, Liberal; R. G. Rate, Halstead; R. H. Robinson, Wichita; R. E. Speirs, Dodge City; J. F. Thurlow, Hays; D. P. Trees, Wichita.

Endowment

C. V. Black, Pratt, Chairman; R. M. Mathews, Overland Park; R. A. Nelson, Wichita; R. J. Ohman, Dodge City; J. L. Perkins, Hutchinson; J. W. Randell, Marysville; R. Schrepfer, Kansas City; F. L. Smith, Jr., Colby; C. C. Underwood, Emporia.

Executive

H. M. Glover, President, Newton; N. L. Francis, President-elect, Wichita; F. E. Wrightman, Past-president, Sabetha; H. St. Clair O'Donnell, First Vice President, Ellsworth; J. C. Mitchell, Sec. Vice President, Salina; J. L. Lattimore, Treasurer, Topeka; Leland Speer, Secretary, Kansas City.

Fee Schedule

W. J. Reals, Wichita, Chairman, Pathology; W. R. Allen, Kansas City, Radiology; H. J. Brown, Winfield, Anesthesiology; J. G. Claypool, Howard, Internal Medicine; G. W. Fields, Scott City, General Parctice; W. H. Fritzemeier, Wichita, Dermatology; L. W. Hatton, Salina, Psychiatry; J. E. Hill, Arkansas City, Ophthalmology; T. C. Hurst, Wichita, Pediatrics; G. B. Joyce, Topeka, Orthopedics; J. G. Kendrick, Wichita, Obstetrics and Gynecology; W. R. Lentz, Seneca, General Practice; W. P. McKnight, Wichita, ENT; P. W. Morgan, Emporia, Internal Medicine; C. R. Openshaw, Hutchinson, Thoracic Surgery; J. G. Phipps, Wichita, General Practice; J. E. Roderick, Salina, Urology; S. L. Vander Velde, Emporia, Surgery.

General Practice Award

B. P. Meeker, Wichita, Chairman; C. W. Bowen, Topeka; F. E. Dillenbeck, El Dorado; R. M. Glover, Newton; L. E. Leigh, Overland Park; W. R. Lentz, Seneca; G. P. Neighbor, Kansas City; A. K. Ratzlaff, Goessel; S. Zweifel, Kingman.

Gerontology

D. V. Preheim, Newton, Chairman; J. O. Austin, Garden City; R. A. Barker, Council Grove; L. J. Beyer, Lyons; J. M. Catlett, Shawnee Mission; T. Dechairo, Westmoreland; D. M. Diefendorf, Waterville; G. M. Edmonds, Horton; H. A. Flanders, Hays; T. S. Gage, Overland Park; J. T. Hamilton, Wichita; A. G. Isaac, Newton; A. M. Isaac, Newton; V. L. Jackson, Altamont; R. McCoy, Coldwater; K. McLain, Ransom; J. J. Marchbanks, Oakley; R. F. Morton, Arkansas City; T. V. Oltman, Riley; J. W. Parker, Burlington; E. S. Rich, Newton; D. L. Rose, Kansas City; H. L. Songer, Lincoln; C. E. Stevenson, Neodesha; G. A. Surface, Ellis; P. W. Thompson, Topeka.

History

R. R. Melton, Marion, Chairman; J. F. Barr, Ottawa; H. C. Clark, Wichita; A. W. Corbett, Emporia; O. W. Davidson, Kansas City; R. D. Grayson, Overland Park; J. G. Hughbanks, Independence; I. A. Koeneke, Halstead; R. H. Major, Kansas City; H. P. Palmer, Scott City; R. A. Schwegler, Lawrence; G. S. Voorhees, Leavenworth.

Hospitals

W. E. Grove, Newton, Chairman; G. B. Athy, Columbus; H. G. Bayles, Fredonia; W. M. Campion, Liberal; M. D. Christensen, Kiowa; J. D. Colt, Manhattan; M. C. Eddy, Hays; D. L. Evans, Manhattan; F. R. Frink, Lawrence; E. R. Gelvin, Concordia; E. T. Gertson, Atwood; J. D. Gough, Chanute; G. F. Gsell, Wichita; R. H. Hill, Meade; C. C. Hunnicutt, Sabetha; G. H. Keene, Wichita; D. B. Parker, Ness City; H. B. Russell, Great Bend; C. D. Snyder, Winfield; E. B. Struxness, Hutchinson; R. E. White, Garnett.

Industrial Medicine

J. F. Lance, Jr., Wichita, Chairman; V. D. Alquist, Baxter Springs; W. L. Anderson, Atchison; N. C. Bos, Hutchinson; J. A. Budetti, Wichita; A. S. J. Clarke, Prairie Village; C. L. Francisco, Kansas City; W. L. Good, Mission; G. S. Hopkins, Topeka; J. J. Hovorka, Emporia; C. A. Isaac, Newton; C. M. Lessenden, Topeka; F. L. Loveland, Topeka; R. H. Moore, Lansing; F. A. Moorhead, Neodesha; A. R. Mueller, Leavenworth; P. C. Nohe, Kansas City; W. F. Powers, Wichita; H. L. Regier, Kansas City; E. C. Sifers, Kansas City; R. W. Urie, Parsons; C. L. White, Great Bend.

Legislation

N. L. Francis, Wichita, Chairman; (Executive Committee plus A.M.A. Delegates).

Maternal Welfare

L. E. Woodard, Wichita, Chairman; A. H. Baum, Dodge City; D. L. Berger, Mission; E. C. Brandsted, McPherson; R. M. Carr, Junction City; H. R. Elliott, Pittsburg; H. M. Floersch, Kansas City; H. M. Foster, Hays; E. S. Gendel, Topeka; D. E. Gray, Topeka; R. G. Heasty, Manhattan; J. G. Kendrick, Wichita; D. S. Klassen, Newton; J. G. Lee, Jr., Kansas City; E. A. Martin, Parsons; O. L. Martin, Salina; C. P. McCoy, Wichita; M. D. Morris, Topeka; F. F. Nyberg, Wichita; E. S. Rich, Newton; W. R. Roy, Topeka; R. Sohlberg, Jr., McPherson; J. C. Schroll, Hutchin-

son; E. F. Steichen, Lenora; D. L. Traylor, Emporia; H. L. Wilcox, Lawrence; D. H. Wood, Pittsburg.

Medical Assistants

H. L. Hiebert, Topeka, Chairman; W. M. Campion, Liberal; C. D. Davenport, Hoisington; C. A. Isaac, Newton; H. P. Jones, Lawrence; S. C. McCrae, Salina; R. H. Moore, Lansing; L. F. Schmaus, Iola; F. L. Smith, Jr., Colby; J. R. Twinem, Olathe.

Medical Economics

K. L. Graham, Leavenworth, Chairman; P. D. Adams, Osage City; E. G. Anderson, Wichita; J. N. Blank, Hutchinson; H. L. Bogan, Baxter Springs; J. K. Griffith, Winfield; G. E. Kassebaum, El Dorado; J. A. McClure, Topeka; M. B. Miller, Topeka; B. A. Nelson, Manhattan; L. S. Nelson, Jr., Salina; C. A. Nystrom, Cawker City; E. B. Scagnelli, Dodge City; R. P. Schellinger, Emporia; F. G. Schenck, Burlingame; R. C. Stanley, Paola; C. H. Steele, Kansas City; T. F. Taylor, Phillipsburg; W. K. Walker, Sedan.

Medical Schools

L. F. Glaser, Hutchinson, Chairman; R. G. Ball, Manhattan; O. W. Davidson, Kansas City; M. C. Eddy, Hays; D. B. Foster, Topeka; N. L. Francis, Wichita; L. G. Glenn, Protection; H. P. Jones, Lawrence; L. C. Joslin, Harper; D. Lukens, Hutchinson; L. F. McKee, Cottonwood Falls; D. Marchbanks, Hill City; B. M. Matassarin, Wichita; J. C. Mitchell, Salina; P. W. Morgan, Emporia; J. G. Phipps, Wichita; M. Snyder, Salina; N. V. Treger, Topeka; J. R. Twinem, Olathe; W. H. Walker, Eskridge.

Medicare

Kansas City Area: J. W. Manley, Kansas City, Chairman; D. L. Berger, Mission; K. L. Graham, Leavenworth; J. G. Lee, Jr., Kansas City; G. J. Pierron, Olathe.

Manhattan Area: C. J. W. Wilen, Manhattan, Chairman; J. D. Colt, Jr., Manhattan; P. H. Hostetter, Manhattan.

Salina Area: L. S. Nelson, Sr., Salina, Chairman; D. A. Anderson, Salina; R. L. Dreher, Salina; N. M. Jenkins, Salina.

Topeka Area: G. B. Joyce, Topeka, Chairman; L. R. Pyle, Topeka; L. L. Saylor, Topeka.

Wichita Area: C. W. Miller, Wichita, Chairman; H. S. Bowman, Wichita; J. G. Kendrick, Wichita; B. P. Meeker, Wichita.

Mental Health

J. A. Grimshaw, Topeka, Chairman; A. J. Adams, Wichita; H. V. Bair, Parsons; A. P. Bay, Topeka; E. P. Carreau, Wichita; I. C. Case, Topeka; O. R. Cram, Jr., Larned; J. A. Dunagin, Topeka; D. E. Eckart, Hutchinson; W. J. Gardner, Halstead; D. C. Greaves, Kansas City; L. W. Hatton, Salina; A. M. Isaac, Newton; G. W. Jackson, Topeka; J. S. Menaker, Wichita; F. C. Newsom, Wichita; W. F. Roth, Jr., Kansas City; W. C. Schwartz, Manhattan; W. A. Warren, Wichita; H. G. Whittington, Lawrence; M. E. Wright, Lawrence.

Necrology

O. R. Clark, Topeka, Chairman; D. E. Gray, Topeka; R. Greer, Topeka; D. Lawson, Topeka; J. A. Segerson, Topeka.

Nominations

T. P. Butcher, Emporia, Chairman; C. M. Barnes, Seneca; C. W. Miller, Wichita; L. S. Nelson, Sr., Salina; H. N. Tihen, Wichita.

Pathology

J. E. Johnson, Kansas City, Chairman; R. J. Eilers, Kansas City; A. A. Fink, Topeka; C. A. Hellwig, Halstead; I. Joffee, Kansas City; W. E. Luedtke, Emporia; W. J. Reals, Wichita; R. J. Rettenmaier, Kansas City; C. J. Weber, Salina.

Perinatal Welfare

W. H. Crouch, Topeka, Chairman; H. Aldis, Fort Scott; R. D. Boles, Dodge City; V. E. Bolton, Kansas City; M. D. Christensen, Kiowa; J. M. Graham, Leavenworth; G. F. Jordan, Jr., Wichita; H. P. Jubelt, Manhattan; R. C. Knappenberger, Wichita; O. L. Martin, Salina; R. E. Pfuetze, Topeka; L. R. Pyle, Topeka; P. T. Schloesser, Topeka; R. N. Shears, Hutchinson; T. E. Young, Topeka.

Postgraduate Study

C. R. Rombold, Wichita, Chairman; W. H. Algie, Kansas City; G. E. Burket, Jr., Kingman; M. H. Delp, Kansas City; D. B. Foster, Topeka; S. R. Friesen, Kansan City; B. C. Gradinger, Halstead; T. W. Graham, Leavenworth; G. C. Hutchison, Hays; D. Lawson, Topeka; D. Lukens, Hutchinson; E. L. Mills, Wichita; E. J. Ryan, Emporia; C. T. Sills, Newton.

Public Policy

F. E. Wrightman, Sabetha, Chairman; C. M. Barnes, Seneca; C. H. Benage, Pittsburg; W. F. Bernstorf, Winfield; T. P. Butcher, Emporia; W. P. Callahan, Wichita; O. W. Davidson, Kansas City; M. C. Eddy, Hays; E. S. Edgerton, Wichita; J. L. Lattimore, Topeka; F. L. Loveland, Topeka; N. E. Melencamp, Dodge City; C. W. Miller, Wichita; B. A. Nelson, Manhattan; L. S. Nelson, Sr., Salina; J. H. A. Peck, St. Francis; G. R. Peters, Kansas City; L. R. Pyle, Topeka; H. N. Tihen, Wichita.

Public Relations

L. S. Nelson, Sr., Salina, Chairman; S. A. Anderson, Clay Center; C. H. Benage, Pittsburg, T. P. Butcher, Emporia; E. W. Crow, Wichita; A. H. Dyck, McPherson; J. L. Lattimore, Topeka; J. W. Manley, Kansas City; G. Marshall, Colby; C. W. Miller, Wichita; R. H. O'Donnell, Ellsworth; L. W. Patzkowsky, Kiowa; J. R. Twinem, Olathe; W. O. Wallace, Atchison.

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From the Stacks

State Medical Library

MRS. BLENDENA EVANS, *Librarian*
Stormont Medical Library, State House
Room 516, Topeka, Kansas
Phone CE 5-0011, ex. 297

Anatomy Monographs available in the library

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Snively, William D. Sea within; the story of our body fluid. Lippincott. 1960.

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White, Abraham. Principles of biochemistry. McGraw-Hill. 1959.

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Albritton, E. C. Standard values in blood. Saunders. 1952.

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Books and periodicals will be sent anywhere in the state. You pay only the postage, four cents for the first pound and one cent for each additional pound.

Interest Growing in Local County Service Benefit Program

Blue Shield's most recent development of a more comprehensive medical-surgical benefit plan, known as the Local County Service Benefit Program, was initiated last March in Butler County under the sponsorship of the Butler County Medical Society. Interest in this plan has been increasing over the state and the program has now been approved by two additional County Medical Societies. McPherson and Montgomery Counties have adopted the program.

McPherson County enrollment began in mid-June and has just been completed. A membership drive in Montgomery County will get under way July 17.

Although it is too early to report enrollment progress in McPherson County, preliminary figures are available from Butler County, revealing successful results. Particular success can be noted in the upgrading to Service Benefits by Blue Cross-Blue Shield Employee Groups formerly carrying Schedules 1 and 2. Over 40 per cent of the total Blue Shield group membership in the county converted to the new program during the campaign in March.

In summarizing the membership gain during the campaign, an estimated 2,360 members in the county are now enrolled in the Schedule 3 program. Overall, Blue Shield membership in all categories was increased approximately 335 members. These results indicate an increasing public desire for more comprehensive programs with predictable coverage.

The Local County Service Benefit Program, frequently referred to as Schedule 3, was developed as a means of providing a greater predictability of coverage for members. In order for the Program to be established in a county, the local Medical Society must first approve it. Then physicians in the area have an opportunity to sign agreements which extend their Blue Shield participation to include the new Local Service Benefit Plan.

To participate in the program, it is necessary for a physician also to be a Blue Shield Participating Physician in Schedule 1 (Plan A) and Schedule 2 (Plan B). On the other hand, the doctor may elect to continue participating in Schedules 1 and 2 without obligation to sponsor the new Service Benefit Program.

The Local County Service Benefit Program is founded on a service basis that removed income as a determining factor. The physician agrees to accept the Blue Shield allowance for covered services according to three conditions:

1. That the patient use the services of a physician participating in the program.

2. That the member has no other surgical-medical coverage.

3. That when hospitalized, the member occupies a semi-private room. Added benefits include first and last day in-hospital medical care; in-hospital consultation allowances; separate fees for assistant surgeons,and a revised surgical allowance scale known as Fee Schedule 3. Schedule 3 increases fees above the Schedule 2 (Plan B) level for the forty most commonly performed surgical procedures. The remaining procedures carry the Schedule 2 allowance.

Questions have been asked concerning the role in the program for non-participating physicians. Listed benefits are available to members, but the special "service benefit" features apply only when services are obtained from physicians participating in the Program.

Service Benefits by physicians participating in only the present Schedules 1 and 2 would be extended only on the income basis already followed under Schedule 2 (Plan B), which is $4,500 or below for family memberships. Allowances would be according to Schedule 3 amounts and would be paid directly to the physician.

Benefits would be available to the member for services obtained from licensed physicians who do not participate in any manner in Blue Shield. These would be paid on an indemnity basis however, and payments would be sent to the members.

Indications point toward continuing interest by other areas in Local County Service Benefit Programs. The first step in developing a local program would be an expression of interest by the local Society to Blue Shield. At this time, committee could be appointed on the local level to work with the Blue Shield staff and investigate the advisability of adopting such a plan in the county.

Committees for 1961-1962

(Continued from page 339)

Relations With Bar Association

G. E. Burket, Jr., Kingman, Chairman; L. G. Allen, Jr., Kansas City; J. O. Baeke, Overland Park; T. R. Hamilton, Kansas City; J. B. Jarrott, Hutchinson; C. S. Joss, Topeka; W. G. Parker, Hoxie; E. J. Ryan, Emporia; J. A. Segerson, Topeka; K. E. Voldeng, Wellington.

Rural Health

C. R. Svoboda, Chapman, Chairman; P. D. Adams, Osage City; V. E. Brown, Sabetha; J. G. Claypool, Howard; F. G. Freeman, Pratt; M. F. Frederick, Hugoton; R. E. Grene, La Crosse; W. A. Harms, Hesston; H. W. Hiesterman, Quinter; P. H. Hostetter, Manhattan; R. L. Krause, Goessel; E. E. Long, Humboldt; R. P. McCarthy, Bethel; D. Marchbanks, Hill City; L. W. Patzkowsky, Kiowa; D. R. Pierce, Topeka; J. G. Rowlett, Paola; J. Scanlon, Horton; M. E. Schulz, Russell; R. R. Snook, McLouth; E. F. Steichen, Lenora; M. H. Waldorf, Jr., Greensburg; H. O. Williams, Cheney; D. H. Wood, Pittsburg; E. D. Yoder, Denton.

Safety

A. C. Eitzen, Hillsboro, Chairman; T. G. Duckett, Hiawatha; J. A. Grove, Newton; C. D. Hensley, Jr., Wichita; A. E. Hiebert, Wichita; J. E. Hill, Arkansas City; C. T. McCoy, Hutchinson; G. R. Maser, Mission; A. L. Nichols, Hiawatha; J. H. A. Peck, St. Francis; R. R. Snook, McLouth; H. E. Snyder, Winfield; H. B. Sullivan, Jr., Shawnee; R. P. Weltmer, Beloit; R. C. Ye, Kansas City.

School Health

C. M. Barnes, Seneca, Chairman; M. D. Athon, Overland Park; W. F. Bernstorf, Winfield; V. L. Branson, Lawrence; J. A. Butin, Chanute; E. J. Chaney, Belleville; W. H. Crouch, Topeka; E. S. Gendel, Topeka; E. D. Greenwood, Topeka; R. H. Greer, Topeka; D. G. Griswold, Newton; J. A. Grove, Newton; R. Hale, Wichita; H. P. Jubelt, Manhattan; M. S. Lake, Salina; O. W. Longwood, Stafford; H. Lutz, Augusta; W. C. Menninger, Topeka; W. E. Myers, Iola; R. A. Nelson, Wichita; R. R. Snook, McLouth; H. R. Wagenblast, Salina.

State Meeting Format

N. L. Francis, Wichita, Chairman; L. H. Leger, Kansas City; J. L. Perkins, Hutchinson; R. K. Purves, Wichita; J. E. Roderick, Salina; E. J. Ryan, Emporia; R. Sohlberg, Jr., McPherson; R. C. Tozer, Topeka.

Stormont Medical Library

B. M. Powell, II, Topeka, Chairman; W. L. Fowler, Concordia; W. E. Hird, Wichita; N. M. Jenkins, Salina; R. C. Lawson, Topeka; G. A. Lessenden, Lawrence; D. S. Lowe, Hiawatha; M. F. Stock, Pittsburg; R. G. Weiner, Halstead; R. P. Woods, Topeka.

Study of Heart Disease

L. E. Peckenschneider, Halstead, Chairman; M. S. Allen, Kansas City; D. R. Bedford, Topeka; J. W. Butin, Wichita; E. W. Crow, Wichita; C. W. Erickson, Pittsburg; L. H. Leger, Kansas City; D. Lukens, Hutchinson; P. W. Morgan, Emporia; R. F. Morton, Arkansas City; G. L. Norris, Winfield; L. F. Schmaus, Iola; C. T. Sills, Newton; H. B. Stryker, Jr., Concordia; D. C. Wakeman, Topeka; C. J. W. Wilen, Manhattan.

The oyster, when the moon is full, opens itself wide, and when the crab looks in he throws in a piece of rock or seaweed and the oyster cannot close again, whereby it serves for food to that crab. This is what happens to him who opens his mouth to tell his secret. He becomes the prey of the treacherous hearer.

—*Leonardo Da Vinci*

We must be prepared during the years ahead to live in a world in which tension and bickering between free nations and the Soviet will be a daily experience.—*Dwight D. Eisenhower*

He that falls in love with himself will have no rivals.—*Benjamin Franklin*

Personalities—IN KANSAS MEDICINE

Dr. Will Menninger, Topeka, led a discussion panel at a session on "The States and the Mentally Ill" June 26 at the Conference of the Governors of the 50 states in Honolulu. Gov. John Anderson was a panelist.

Dr. Wm. J. Reals, Wichita, was a Judge at the National Science Fair in the division of "Pathology-Medical Technology Awards" which was held in Kansas City, Missouri, May 10-13.

Dr. Dale W. Anderson, Augusta, presented the lesson to the Science Seminar students May 4. His topic was "Cancer."

Dr. Robert Wallerstein, Topeka, has been elected chairman of the committee on research of the Group for the Advancement of Psychiatry.

Dr. Ernest P. Carreau, Wichita, attended the American Goiter Association meeting in Philadelphia, May 3-6.

Dr. J. Gordon Claypool, Howard, attended the 42nd annual convention of the American College of Physicians, held at Miami, Florida, the middle of May. Dr. Claypool was one of the more than 300 who received his advancement to Fellowship.

Dr. Bernard Hall, Topeka, has been named to a three-year term on the committee on the history of psychiatry of the American Psychiatric Association. Dr. Hall spoke on "Your Investment in Mental Health" at a public lecture at Donnelly College in Kansas City, Kansas.

Dr. Mary J. Blood, Wichita, attended a Symposium on Pediatrics at the University of Kansas Medical Center, May 6-8.

Drs. Joseph Stein and **John Segerson,** Topeka, attended a meeting of the American Academy of Neurology in Detroit. Dr. Segerson was a leader of a one-day training program in Topeka for Kansas Catholic Lay Volunteers who will work in South America.

Dr. Ralph L. Drake, Wichita, attended the American Academy of Neurology meeting in Detroit, April 24-29.

Dr. Karl Menninger, Topeka, spoke on "Changing Viewpoints in Criminology" at the 60th anniversary dinner of the John Howard Association in May in Chicago. The association is a private group which studies and makes recommendations on penal reform and criminology.

Dr. E. W. Christmann, Wamego, has announced Dr. Wm. Braden has associated with him as of July 1.

Dr. Chas. LeRoy Williams, a Wichita cardiologist, was named president of the Kansas Heart Association during its 12th annual convention on May 27.

NEW MEMBERS

The JOURNAL *takes this opportunity to welcome these new members into the Kansas Medical Society.*

Grady N. Coker, Jr., M.D.
Gelvin-Haughey Clinic
Concordia, Kansas

Tex E. Fury, M.D.
1624 Delaware
Lawrence, Kansas

Eugene McCormick, M.D.
Conway Springs, Kansas

William J. Porter, M.D.
American Independent Oil Co.
P. O. Box 69
Kuwait, Arabia

Donald E. Sklenar, M.D.
Wellington, Kansas

Roger L. Youmans, M.D.
K.U. Medical Center
39th & Rainbow Blvd.
Kansas City 12, Kansas

KMS Obituaries

ROBERT C. GRIBBLE, M.D.

Dr. Robert Gribble, 48, Dodge City, died June 1 at the University of Kansas Medical Center.

Dr. Gribble was born in 1912. He graduated from the University of Kansas School of Medicine in 1937 and interned in Santa Barbara, California where he was married in 1938. He had practiced medicine in Dodge City since 1946 following his release from World War II service as a medical officer with rank of major.

Survivors are Mrs. Gribble; a daughter, Karen; a son, Robert; three sisters and five brothers.

CHARLES N. JOHNSON, M.D.

Dr. C. N. Johnson, 75, Wichita, died May 26 in St. Francis Hospital in Wichita. He was born in 1885 near Andale. In 1910 he graduated from Northwestern University Medical School. He interned at St. Francis Hospital, Wichita, and spent three years practicing at Mount Hope and moved to Wichita in 1917. He married Mable Allison in 1912 in Wichita. He served as captain in World War I and as major in World War II.

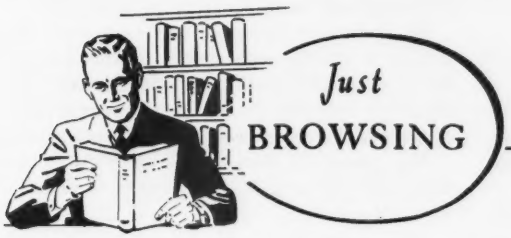
Survivors include Mrs. Johnson, three sons, a daughter, one brother and twelve grandchildren.

JUSTIN J. McDONALD, M.D.

Dr. McDonald, 69, who practiced in Coffeyville nine years before moving to Bartlesville recently, died May 16.

He was born in 1893 in Arlington, Illinois and in 1914 he graduated from the University of Illinois Medical School. In 1926, in North Dakota, he married Miss Avis Pillsbury. Dr. McDonald was an Eye, Ear, Nose and Throat specialist.

He is survived by Mrs. McDonald, a daughter, a son, three sisters, one brother, and six grandchildren.



With the centennial of our state and the centennial of the Civil War both coming during this year, Kansans seem more "centennial conscious" than others, and along with the growing of beards and wearing of hoop skirts and bonnets there has come the production of some commenorative publications. One of these recently off the press has come to attention at the end of the school year. *The Doctor, 1861-1961,* is a 32 page loose-leaf book, bound with plastic rings, and compiled by some members of the class of 1961 at KUMC.

Having had considerable experience in assembling such documents in the preparation of the KMS Centennial issue of the JOURNAL two years ago, I can appreciate the efforts which have been put into this interesting book. Introduced as a supplement to the Yearbook, it has attracted considerable attention and drawn favorable comment from those who have seen it.

The book traces, principally through pictures and appropriate captions, the highlights of Kansas medicine from Territorial days to the present. The part which was played by physicians during the days of "Bleeding Kansas" is amazing, as they took their places in the struggle between slavery forces and abolitionists—and on both sides. The border war, and finally, the drafting of the adopted Wyandotte Constitution with Kansas' admission to the Union saw medical men in positions of prominence.

The impact of the Civil War struck Kansas during the state's infancy and the medical men of Kansas contributed their share and more—even including one Major General in command of the Army of the Frontier.

Early statehood days, Carrie Nation, the Spanish-American War, and the early days of the School of Medicine at the University of Kansas are all portrayed, as well as Dr. Crumbine's efforts in the field of Public Health. The Brinkley affair, without which no historical treatise of Kansas would be complete, receives due attention.

Appropriately, the emphasis is on a tribute to the men who did so much to help develop the medical school at the University of Kansas—both those who got it started as a two-year plan, and those who later helped to get approval for the four-year school and develop it to its present level of prestige. There have been many worthy men engaged in this effort, and it is fitting that they should be honored by the current publication.

The little book is well done, is interesting material, and is worth the efforts which the publishers expended to bring it into being. It should take its place among the preserved documents of Kansas medicine.

Additional copies are available in limited numbers, at a cost of $2.00, and may be ordered from John B. Runnels, M.D. or George F. Sheldon, M.D., University of Kansas Medical Center.—O.R.C.

Official Proceedings

(Continued from page 336)

fails to adequately prepare the young physician to do general practice in his own community, and

WHEREAS, each segment of organized medicine has—and still does—determine the minute details and over-all content of their respective training programs, therefore

Be It Resolved that the Council on Medical Education and Hospitals of the American Medical Association be requested to formulate other pilot two-year progressive training programs which are acceptable to the American Academy of General Practice, the only national association representing general practice.

RESOLUTION NO. 59

WHEREAS, a committee of the Kansas Medical Society authorized by a Special House of Delegates meeting has been meeting as part of a committee appointed by the Governor of Kansas and has produced a plan to make available medical services to the persons receiving old age assistance or indigent care, and

WHEREAS, KPS has worked closely in cooperation with its sponsoring body, the Kansas Medical Society, in helping the Kansas Medical Society provide medical services for the people of Kansas, and

WHEREAS, the Kansas Medical Society wishes to sponsor medical services for those persons on OAA at the highest efficiency and at lowest possible cost, and

WHEREAS, it would materially increase the cost of such a plan if a new bureau was to be formed, personnel secured and equipment procured, and

WHEREAS, Blue Cross-Blue Shield has the necessary skill, personnel and equipment for providing these services of fiscal agent on a non-profit basis

*Be It Resolve*d that the Kansas Medical Society strongly urge the Governor of Kansas that Kansas Blue Cross-Blue Shield be designated the fiscal agent if and when such program is instituted.

RESOLUTION NO. 60

Reno County Medical Society (1)

WHEREAS, the Kansas State Board of Health has become increasingly active in fields which might be interpreted as properly belonging to the practice of private medicine, and

WHEREAS, the component county societies of the Kansas Medical Society are frequently in ignorance of the details regarding proposed surveys, studies and clinics planned by the Kansas State Board of Health, therefore

Be It Resolved that the most effective manner of operating public health surveys, studies and clinics will be with the informed and cooperative support of the component medical society involved, and

Be It Further Resolved that subsequent surveys, studies or clinics contemplated by the Kansas State Board of Health shall be conducted only after the expressed approval of and in such manner as is acceptable to the component medical society involved.

The president, then, called upon the spokesmen from each of the districts for which new councilors were to be elected:

District No. 2—J. W. Manley, M.D., re-elected to another three-year term.

District No. 4—Dick B. McKee, M.D., re-elected to another three-year term.

District No. 11—William J. Reals, M.D., re-elected to another three-year term.

District No. 13—A. M. Cherner, M.D., re-elected to a three-year term.

District No. 14—The committee reported they would caucus later and send the name of their councilor to the Executive Office by mail.

District No. 15—Evan R. Williams, M.D., Dodge City, was elected to a three-year term.

The president then declared Dr. Harold M. Glover to be the new president of the Kansas Medical Society, who took the chair and adjourned the House of Delegates at 3:55 p.m.

The Kansas Press Looks at Medicine

(Continued from page 330)

enthusiasm for it. The need may not be there, but if sufficient numbers can be convinced it is good and they respond by cheering, the proponents can say they are making political hay.

This political approach is difficult for the medical profession to cope with. Given a little time the doctors just might swing this tide in another direction.—*Arkansas City Daily Traveler,* May 18, 1961.

From the Stacks

(Continued from page 340)

Asimov, Isaac. The living river. Abelard-Schuman. 1959.

Ciba Foundation. Peripheral circulation in man. Little, Brown. 1954.

Diggs, Lemuel. The morphology of human blood cells. Saunders. 1956.

Roth, G. M. Tobacco and the cardiovascular system. Thomas. 1951.

Altman, P. Handbook of respiration. Saunders. 1958.

Duncan, G. G. Diseases of metabolism. Saunders. 1955.

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1. Blank, P., and Boas, H.: Improved analgesia for moderate pain, Ann. West. Med. & Surg. 6:376, 1952. 2. Bonica, J. J., et al.: The management of postpartum pain with dihydrohydroxycodeinone (Percodan): Evaluation with codeine and placebo, West. J. Surg. 65:84, 1957. 3. Cass, L. J., and Frederick, W. S.: A controlled study in pain relief, M. Times 84:1318, 1956. 4. Chasko, W. J.: Pain-free dental surgery: Postoperative extension of the pain-free state, J. District of Columbia Dent. Soc. 31:3, No. 5, 1956. 5. Cozen, L.: Office Orthopedics, ed. 2, Philadelphia, Lea & Febiger, 1953, pp. 120, 138, 145, 156, 234. 6. Nicolson, W. P., Jr., and Skandalakis, J. E.: Control of postoperative pain, J.M.A. Georgia 46:471, 1957. 7. Piper, C. E., and Nicklas, F. W.: Percodan for pain in industrial practice, Indust. Med. 23:510, 1954; abstracted, Clin. Med. 3:1008, 1956, Current M. Digest 22:135, No. 3, 1955.

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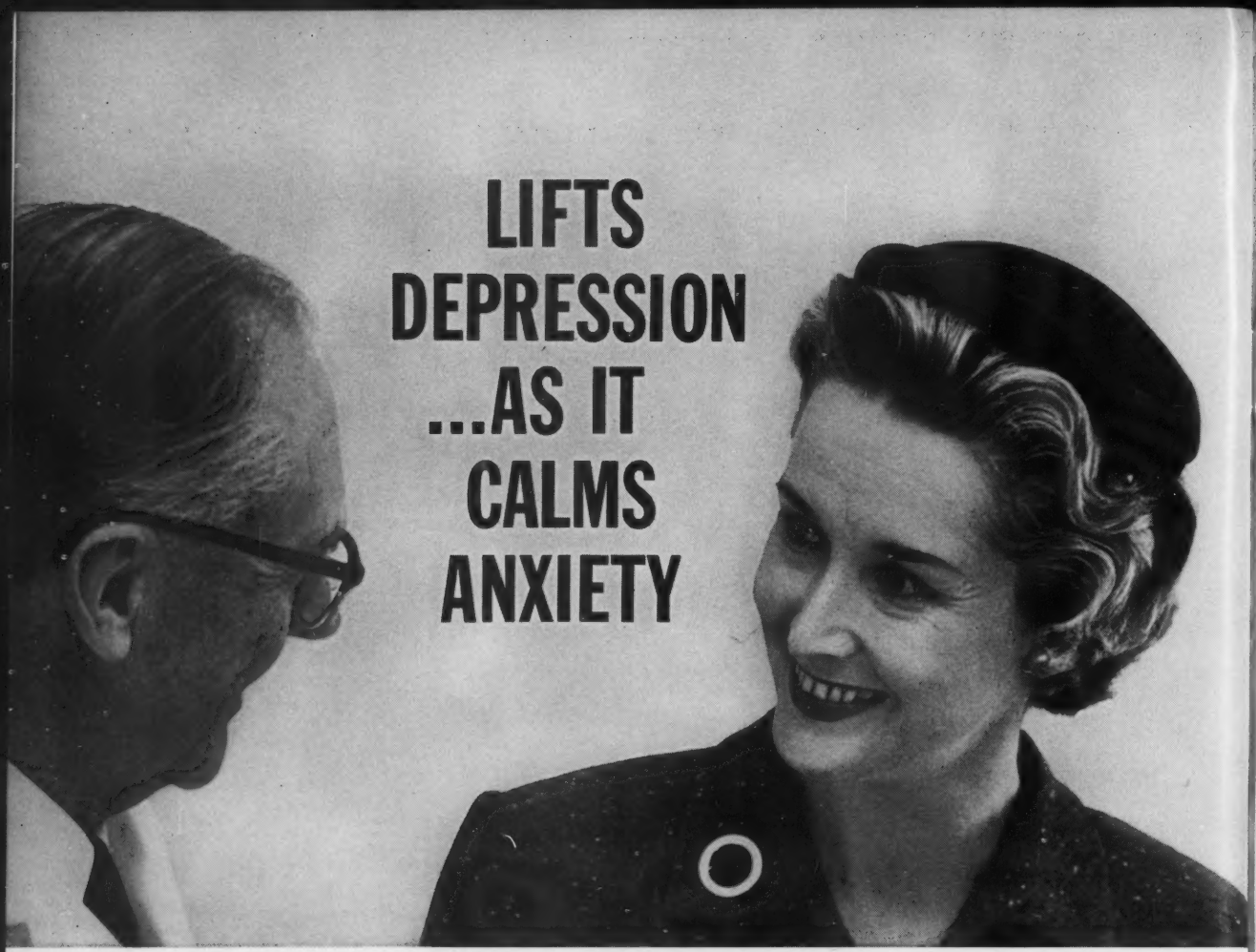

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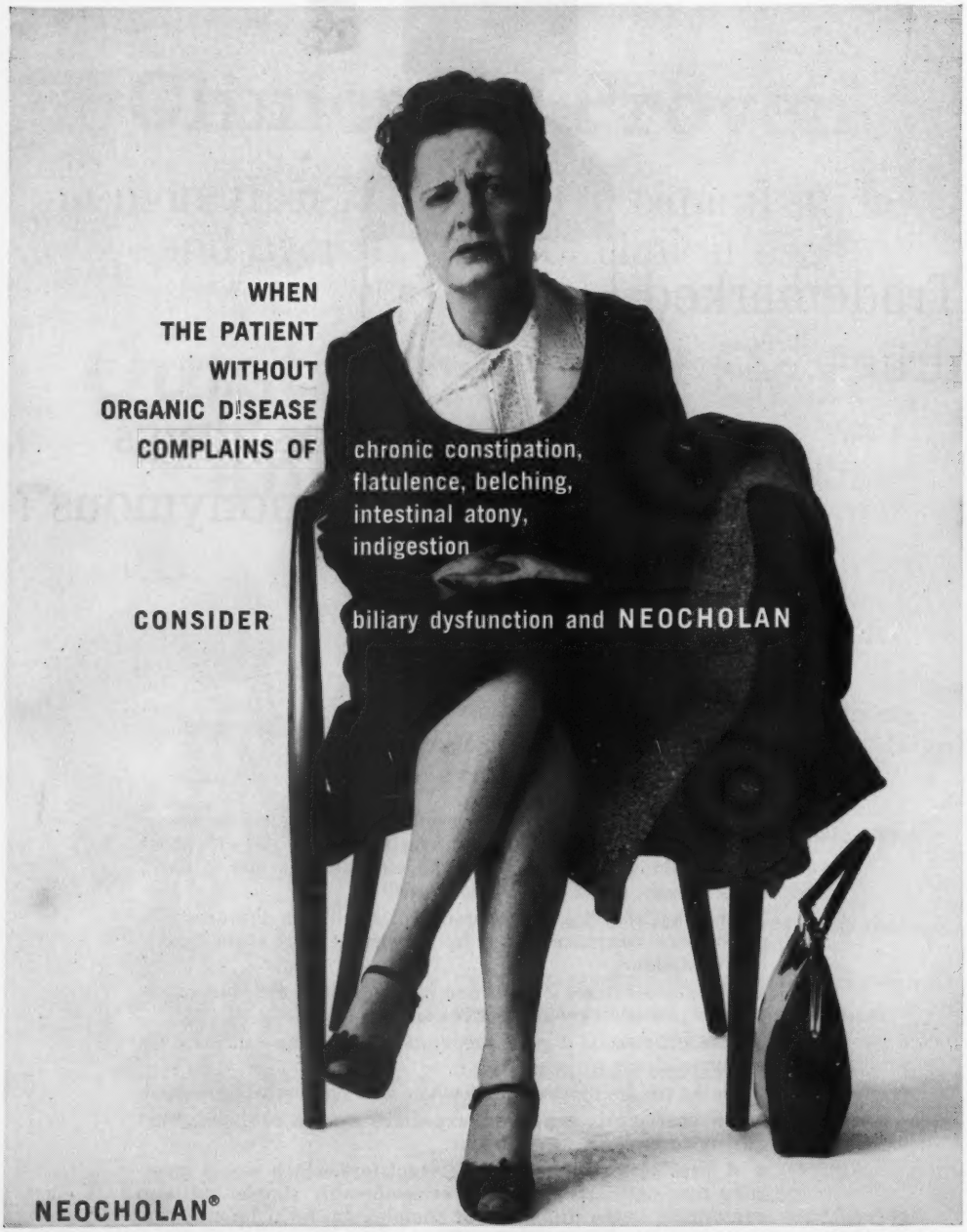

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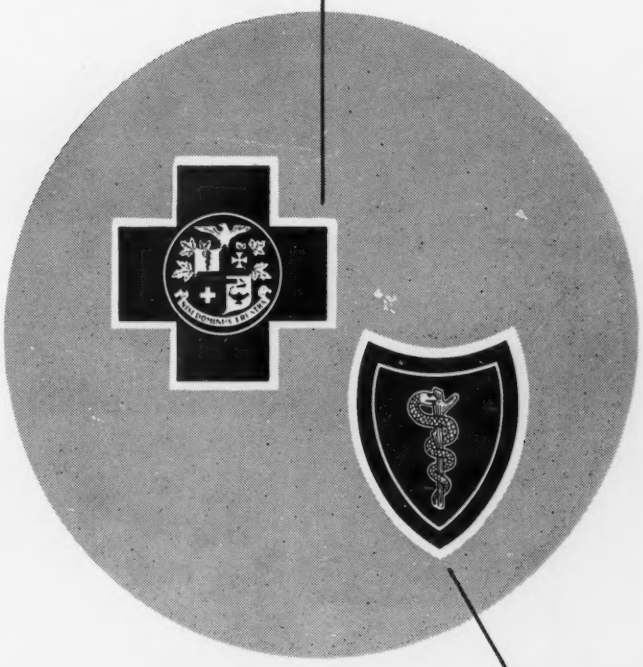

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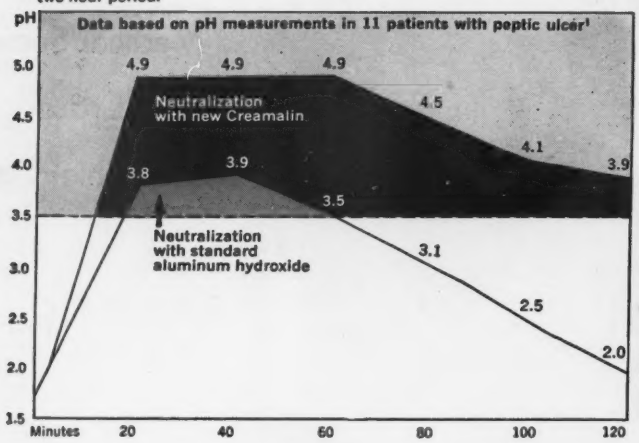

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1. Data in the files of the Department of Medical Research, Winthrop Laboratories. 2. Hinkel, E. T., Jr.; Fisher, M. P., and Tainter, M. L.: J. Am. Pharm. A. (Scient. Ed.) 48:384, July, 1959.

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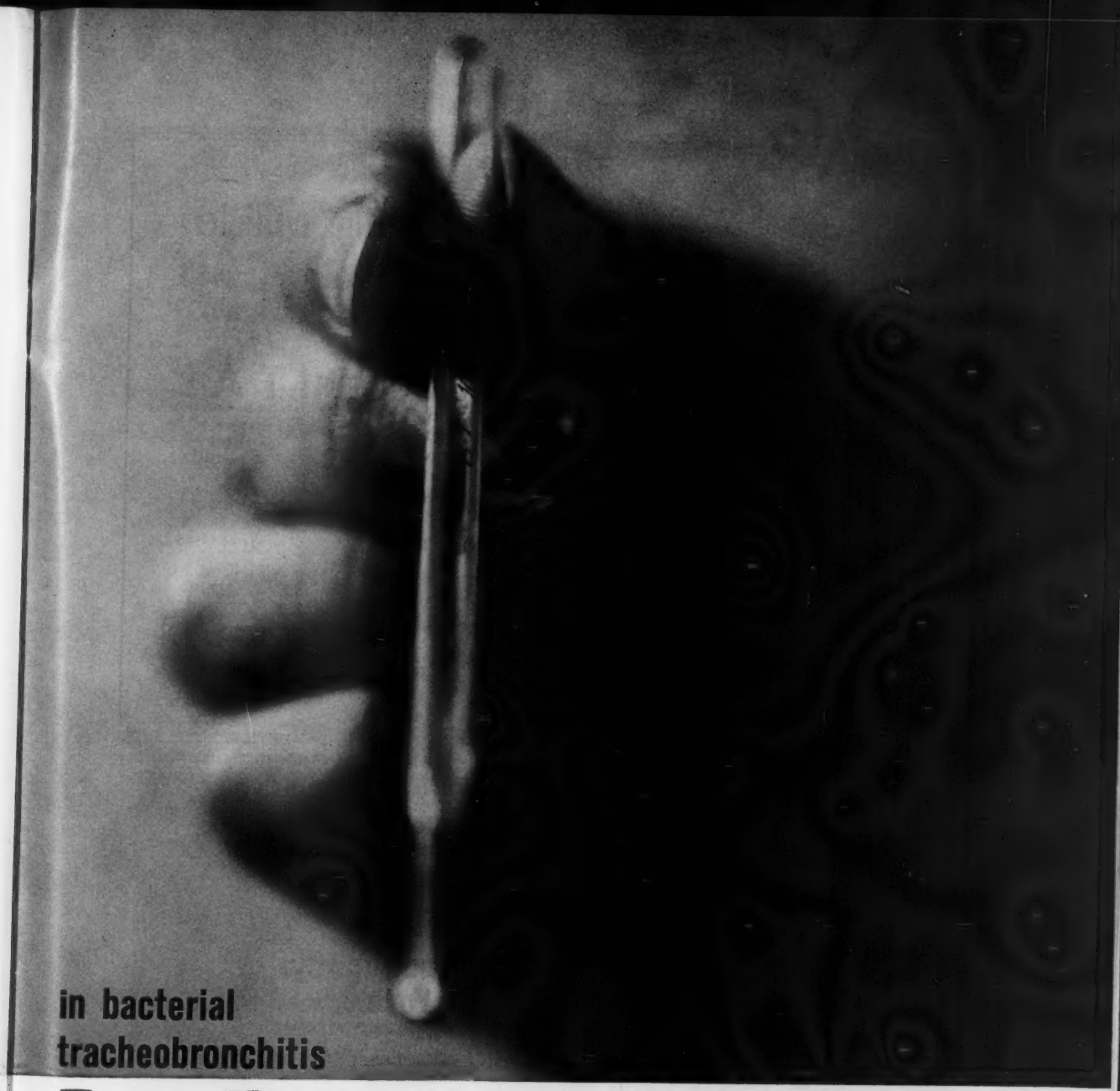

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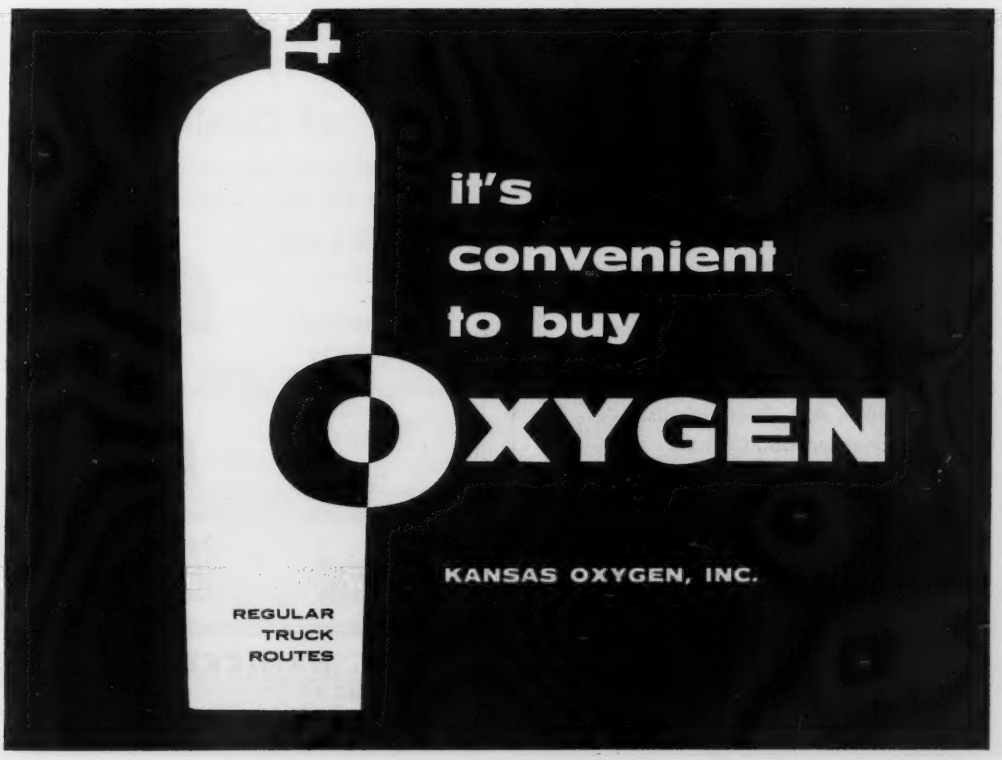

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(1) Danowski, T. S.: Diabetes Mellitus, Baltimore, Williams & Wilkins, 1957, p. 239. (2) McCune, W. G.: M. Clin. North America 44:1479, 1960. (3) Ackerman, R. F., et al.: Diabetes 7:398, 1958.

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earlier detection of peripheral vascular disease key to improved therapeutic response

In practically all peripheral vascular disease cases where marked occlusion with severe ulceration or frank gangrene has not developed, patients can be assured that excellent treatment is available and many symptoms can be relieved.[1] Routine palpation of peripheral pulses[2] and performance of clinical tests for peripheral arterial disease[3] will help earlier diagnosis. Consequently treatment can be instituted sooner, improving likelihood of a favorable response to therapy.

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increases deep peripheral circulation by direct action ...without troublesome side effects

VASODĪLAN'S record of safety and effectiveness in the management of peripheral vascular disease has been established clinically.[4-6] Clarkson and Le Pere report: With strictly a clinical office approach, isoxsuprine [VASODĪLAN] was used in the treatment of 100 patients with peripheral vascular disorders. Definite clinical improvement was obtained in 89 per cent of these patients."[5] They further state: "In particular, the symptoms of pain, cramping, numbness, and cold were consistently relieved."[5]

Contraindications—There are no known contraindications to oral administration of VASODĪLAN in recommended doses.

Cautions—VASODĪLAN should not be given immediately postpartum or in the presence of arterial bleeding. Parenteral administration is not recommended in the presence of hypotension or tachycardia. Intravenous administration is not recommended because of the increased likelihood of side effects.

Side effects—Few side effects occur when given in recommended doses. Occasional palpitation and dizziness can usually be controlled by dosage adjustment. Single intramuscular doses of 10 mg. or more may result in hypotension or tachycardia.

Dosage and administration—Oral—10 to 20 mg. (1 to 2 tablets) t.i.d. or q.i.d.; I.M.—5 to 10 mg. b.i.d. or t.i.d.

Supplied—10 mg. tablets, bottles of 100; 2 cc. ampuls (5 mg./cc.) for intramuscular use, boxes of 6.

For complete details on indications, dosage, administration and clinical background of VASODĪLAN, see the brochure of this product available on request from Mead Johnson Laboratories, Evansville 21, Indiana.

References: (1) Lieberman, J. S.: GP *21*:133-143 (March) 1960. (2) DeWeese, J. A.: New England J. Med. *262*:1214-1217 (June 16) 1960. (3) Winsor, T.: Peripheral Vascular Diseases: An Objective Approach, Springfield, Illinois, Charles C Thomas, 1959, pp. 457-458. (4) Kaindl, F.; Samuels, S. S.; Selman, D., and Shaftel, H.: Angiology *10*:185-192 (Aug.) 1959. (5) Clarkson, I. S., and Le Pere, D. M.: Angiology *11*:190-192 (June) 1960. (6) Samuels, S. S., and Shaftel, H. E.: J.A.M.A. *17*:142-145 (Sept. 12) 1959.

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